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ROYAL SOCIETY OF MEDICINE

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JOHN NACHBAR, M.A., M.D.
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THE EDITORIAL COMMITTEE

VOLUME THE SECOND

SESSION 1908-9

PART III

ODONTOLOGICAL SECTION	OTOLOGICAL SECTION
PATHOLOGICAL SECTION	SURGICAL SECTION
THERAPEUTICAL AND PHARMACOLOGICAL SECTION	

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COMPRISING THE REPORT OF THE PROCEEDINGS FOR THE
SESSION 1908-9

ODONTOLOGICAL SECTION



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1909

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The Council think it right to state that the Society does not hold itself in any way responsible for the statements made or the views put forward in the various papers.

Odontological Section.

October 26, 1908.

Mr. H. BALDWIN, Vice-President of the Section, in the Chair.

An Amalgamator—a Mechanical Device for Amalgamating Alloys.

By W. FRANCIS MELLERSH.

It has been suggested to me the subject of this note might form a suitable communication to bring before this Section of the Royal Society of Medicine.

The late Mr. Thomas Fletcher was, I believe, the first to draw attention to the advantages of amalgamating alloys for fillings by shaking in a glass tube. It is claimed for amalgam made in this manner that more perfect amalgamation is effected, it being impossible for particles of metal to become burnished, as may occur when force is applied in a mortar. This and other methods, however, involve a certain expenditure of time on the part of the operator or his assistant. My desire was to obtain mechanically a result hitherto, so far as I am aware, possible only by hand.

The apparatus designed consists of a clockwork motor, which imparts movement to a pendulum carrying a small glass tube, this movement being as much as possible like that of the hand and wrist when shaking a tube. This apparatus I term an amalgamator, and it has proved most useful in practice, for by its aid a perfect mix may be obtained ready at any desired time.

The proportions of alloy and mercury being weighed, or measured with Mr. W. J. May's apparatus or Tulloch's spoon, are placed in the tube and the motor started. An operator, knowing how long the particular make of alloy he prefers takes to amalgamate, may have this

ready for use by the time the preparation of the cavity is completed. The mix is sufficiently amalgamated when it has the appearance of coarse gunpowder, but if left sufficiently long can be worked up into a ball.

This apparatus is merely an experimental one, and the case unnecessarily large. A more compact one could be easily produced, and attachments might be made for the electric engine, lathe, or water motor. I venture to hope such apparatus may be found useful as a time-saver, and, being an instrument of precision, suitable in all cases where it is desired to carry out experimental work, as by its aid alloys may be amalgamated at a definite speed and for a definite time.

The CHAIRMAN (Mr. H. Baldwin) said Mr. Mellersh had produced another ingenious invention, which he himself had had the pleasure of seeing before, and it certainly seemed to do its work extremely well. It was a great advantage to be able to save time, even a few seconds, when the operation was one that recurred frequently during the day. Instead of having to shake the amalgam in the tube by hand it was a convenience to be able to do it mechanically.

The Teeth of Early Man.

By WILLIAM WRIGHT, F.R.C.S.

[ABSTRACT.]

THE physical remains of Man are first met in strata of the Mid-Palæolithic Period—the age of the mammoth. Specimens of his teeth are forthcoming from the Spy Caves, from caves at Krapina and Mentone, from deposits at Naulette, Brünn, and Galley Hill in Kent.

The teeth of the Spy jaws are characterized by the oblique insertion of the incisors, by the bifid roots of the premolar teeth in one of the two specimens (Spy II), and by the relative large size of the third molars, which were not sensibly smaller than the first or second. The teeth of the Naulette jaw conform in this last particular to those of the Spy jaws. As to the Krapina teeth, an attempt has recently been made by Adloff to separate Krapina man from present-day man by reason of alleged differences in the teeth. The chief difference lies in the fusion and prismatic conformation of the roots of the molar teeth. It was found that out of twenty-three upper molars only two had three separate roots, out of twenty-four lower molars only five had two separate roots. In this respect the teeth of Krapina man differed widely from the teeth of Spy man, in whom the roots were separate and distinct. The view of Adloff has, however, been strongly opposed by Gorjanovic-Kramberger, who has shown that the fusion and peculiar conformation of the roots are not confined to Krapina man, but can be observed in the teeth of living man. The relatively large size of the third molar in the Spy jaws is a condition by no means constant in the Krapina jaws, for in several specimens there is a marked diminution in the size of the third molar when compared with the first and second molars.

The Mentone teeth, which have attracted most attention, are those of an old woman and a boy, which were found in La Grotte des Enfants, under 10 metres of stalagmitic deposit in strata of Mid-Palæolithic date. They are the earliest remains of man from the Mentone Caves. The youth's teeth were remarkable because of the great length of the molar series and because of the quincusp nature of the crowns of all the lower molars. In these respects an approach is made to the teeth of negroes, an approach which, it is interesting to note, is

confirmed by certain features of the cranium. The skull, including the jaws and teeth found at Brünn, was normal in its dentition, except for the fact that the third lower molar slightly surpassed in length the second.

The Galley Hill skeleton was found by Mr. Robert Elliott, of Camberwell, in stratified palæolithic gravel 8 ft. below the surface and 2 ft. above the chalk. The only feature of interest in the dentition is the size of the third molar, which, as in the Spy specimens, was not sensibly smaller than the first or second.

It will thus be seen that the teeth of the earliest man whose remains are known to us are practically identical with those of men living to-day. The third molar was probably a little longer, but even then there were exceptions, as for instance, the Krapina jaws. The frequency with which the roots of the molars in these jaws (Krapina) were fused into a prismatic mass is to be attributed to individual variation and hereditary transmission. The teeth of mid-palæolithic man were, on the whole, good, but the jaws of the old woman from the Mentone Cave were almost edentulous.

Passing to the remains of man from the Late Palæolithic Period, the age of the reindeer, reference is due to the teeth of the men found at Cro-Magnon and at Chancelade. As to Cro-Magnon man the most interesting feature of his dentition was the small size of the third molar, as the most striking feature of the jaws of Chancelade man was the large number of teeth which had been lost, for, with the exception of the right lateral incisor, there was no tooth in the upper jaws. The teeth had, with the possible exception of the canines, been undoubtedly lost during life. Teeth were not, therefore, invariably good in those days.

In neolithic time a condition not infrequently found was absence of the whole lower molar series; it was probably attributable to food packing. Retraction of the gums and erosion of the dentine at the neck of the tooth were apparently not uncommon. Two instances of teeth congenitally missing are deserving of mention—one from East Yorkshire, in which an upper lateral incisor was absent, the other from Mount Batten, near Plymouth, in which both lateral upper incisors were wanting. The only instance of an accessory tooth was a fourth upper molar found in a skull from an Early Iron Age burial at Driffield.

The teeth of early man were frequently worn down to the neck. The condition was probably due to the mixture with the corn of grit from the stone querns.

Two of the most perfect sets of teeth shown upon the screen were not, properly speaking, of prehistoric date at all. They were the teeth of a Roman centurion and a Roman lady, preserved in the York Museum.

DISCUSSION.

Mr. F. J. BENNETT desired to ask a question in connexion with the bite. The author suggested that the articulation of the teeth was not an interdigitating one, but one in which the teeth met completely tooth to tooth. It was difficult to know how such a condition could take place in young teeth; as long as the cusps were in developmental condition, it was almost impossible that they could meet in the way described. After the teeth were worn down into flat surfaces they might come one upon the top of the other, but while the cusps were present it appeared to him they must interdigitate, unless it was assumed that they were developed without cusps, an assumption which it was impossible to make. It was not at all impossible to imagine that after a good deal of wear and tear had taken place, tooth might meet tooth. With regard to the cases of erosions or pyorrhœa, or whatever it was that laid bare the necks of the teeth, he wished to know whether the author had made a microscopical or sectional examination of the teeth and the alveolus. In some specimens he himself showed at the British Medical Association meeting in Exeter last year he was able to exhibit an ancient skull, the teeth of which had large cavities, which at first sight seemed to be due to erosion or caries, so much so that one or two well-known members of the Society thought at first they were caused by decay. There was no doubt, however, that it was a post-mortem condition. When sections of the teeth were made, quite a typical boring of the teeth was found, due to a post-mortem perforation by some insect, or the larvæ of an insect. Therefore he wished to know whether there was any possibility of a microscopical examination being made of any of the teeth which seemed to suggest pyorrhœa or erosion. In the specimen he showed at Exeter a curious condition was that the bony socket was actually, in some cases, pushed up, and seemed to be thickened, but on cutting a section it was seen to be a thin layer of bone which had been arched over and led into a cavity. There was no question but what that was a post-mortem condition, as was freely acknowledged by the many experts who saw it. It would lead a little further on the road if it were known whether there was any possibility of any part of the condition shown being a post-mortem condition. His own impression was that a vast number of ancient skulls, if examined microscopically, would be found to be riddled, although there was not the slightest appearance in some of the teeth until a section was made. In other cases, the neck of the tooth was found largely eaten into, and gave rise to the supposition that either caries or some inflammatory condition had been present.

Mr. C. ROBBINS said the author had gone back in a most able manner through many ages, but had somewhat disappointed him with regard to palaeolithic man. His own impression was that palaeolithic man would have larger jaws and finer teeth. He had been interested in the point with regard to the wisdom tooth being larger than the other two molars. In some photographs of specimens he had shown with regard to the Harlyn Bay discovery, the third molars were quite as large in some cases, if not larger; but he did not think it was possible to make any special deductions, because in practice one often found that even to-day the wisdom tooth, especially an upper wisdom tooth, varied in size. About the largest tooth he ever saw was an upper wisdom tooth. The ground surface of the tooth was very evident in many specimens shown on the screen, and was what one would expect to find; but he did not think any deductions could be drawn, because the same condition was shown in our early Saxon forefathers. The specimens from Hythe, in Kent, showed quite as much grinding down, and even in private practice one had been sometimes confused by seeing patients whose food would be of the modern kind, and yet an immense deal of attrition of the enamel surface on the crowns was evident. He had sometimes imagined that this condition was associated with gouty affections, but was unable to say for certain. He had also been very much interested in the skull in which the lateral was missing. His impression had always been that the missing lateral was a phenomenon of modern times—that the lateral teeth were gradually being crowded out; but if a case could be traced as far back as palaeolithic man it seemed that the process was going on very slowly. With regard to the skull in which a hole was shown in the temporal bone, he wished to know whether that was a post-mortem cavity, because if so it accorded with the suggestions made with regard to most neolithic burials, that there was generally a hole made in the temporal bone, presumably to let out the spirit on its journey. He had with him one or two slides which he would like to show to illustrate the point with regard to the third molar being larger than the second. The first slide showed one of the skulls of the Harlyn Bay discovery. It was found at a depth of 17 ft. below the sand, and was supposed to have belonged to the late Neolithic Period. It would be seen that the third molar was as large as, if not larger than, the first or second molars. The second slide showed another skull from the same depth and of the same period, and as far as could be seen the third molar was quite as large as the other two. The next slide showed a mandible discovered at the same time and in the same place, but the third molar was not as large. He would be interested if the author could say whether the skeletons were found bent or in the ordinary position.

The CHAIRMAN (Mr. H. Baldwin) said there was something very fascinating in examining the skulls and teeth of primitive man, because of the naturally great desire to know, as far as possible, what was our ancestry, and what our earliest progenitors were like. It was pleasing to learn that man at the present day, so far as his teeth were concerned, and as compared with primitive man, was not so degenerate as we had been prone to think, especially in connexion

with missing lateral incisors, absorption of the alveolar process, possibly due to pyorrhœa, the small third molar, and the presence of caries. He thought the excavations on the buccal side of some of the molars, as seen on the screen, were probably due to some form of slow caries; it looked more like that than a post-mortem effect. He would be glad if the author could say something about the cerebral capacity—whether the cerebral capacity of palæolithic man compared at all with that of the average European at the present time. The post-mortem changes to which Mr. Bennett had referred were generally in the form of holes drilled through the teeth in various directions. Mr. Tomes drew attention to the matter some years ago. The teeth were sometimes found to be drilled through and through by some organism after burial. The cavities shown in the slides, however, appeared to be more of the nature of slow caries; if palæolithic man had been in the habit of using a toothbrush, they might have been put down as toothbrush friction grooves. There was no doubt, however, in his own mind that the grooves were due to slow caries.

Mr. LEWIN PAYNE thought most of the members would be surprised to note the apparent prevalence of pyorrhœa, caries, and irregularity in regard to the position and number of teeth in connexion with palæolithic man, and he would like to know whether the author could give any idea as to the relative frequency of these conditions in the specimens he had examined. With regard to the blending of roots, he would like to know whether there was any possibility of that being due to a pathological or post-mortem change, and whether sections had been taken showing the structures in situ.

Mr. JAMES asked on what the author based his statement that the third molar is smaller than the first or second molar in modern times—whether a large number of measurements had been made from skulls.

Mr. ROWNTREE thought the effects shown in the slides were those associated with pyorrhœa and marginal caries. There was no caries of the crown of the teeth, and he understood that, at the present time, pyorrhœa was very common amongst omnibus horses and domestic animals. It was quite conceivable that possibly some similarity of food or conditions existed. He supposed that the condition of life of the London omnibus horse was very much more similar to that of prehistoric man than was the life of civilized man to-day. Another point in which he was particularly interested was the absence of those irregularities of the teeth and of the dental arch usually associated with the presence of adenoids and mouth breathing, which one had always believed were more or less modern conditions. He would like to know whether the author had met with any instance of such defect or abnormality in the shape of the jaws as had led him to believe that it might result from the presence of adenoids, mouth breathing, or similar conditions.

Dr. WRIGHT, in reply, felt inclined to accept the suggestion that when the teeth were first erupted they did not lie over each other, but that the upper teeth lay over the interval between the lower teeth, and that as wearing took

place the bite occasionally shifted. He had made no sections of teeth, but he thought that in some of the specimens he had shown there could be no doubt at all that the conditions were not due to post-mortem changes. The effects of inflammatory processes could be observed. He was interested to hear that the upper wisdom tooth was still sometimes very large. He had no reason, other than the fact that all text-books seemed to be in agreement, for stating that the third molar, at the present time, was much smaller than the first or second. The missing incisor was not found in palæolithic man, but in neolithic man. Bad teeth and small third molars were occasionally found in palæolithic man. With regard to the Harlyn Bay discoveries, it should be mentioned that not all the skulls in the Harlyn Bay Museum were found at Harlyn Bay. Further, the cemetery at Harlyn Bay seemed to belong to the Early Iron Age (about 1000 B.C.). With regard to the method of burial, it was almost invariable, throughout prehistoric time, to bury the dead in a crouching position. Now and then, however, bodies were found buried in a straight position. With regard to the cerebral capacity, on the whole the cerebral capacity of palæolithic man seemed to have been a little smaller than that of living man; but one had to be very chary in making any assertion, because palæolithic skulls were few in number and mostly fragmentary—the latter fact militating against any precise estimation of the cranial capacity. One of the skulls, however, was a skull that had a cranial capacity which would be almost extreme at the present time, viz., the Chancelade skull, which had a cranial capacity of 1,710 c.c. With regard to the frequency of pyorrhœa and caries, the general impression he gathered from the skulls was that caries was more common to-day than it was then, but he had no statistics to lay before them. The coarse food of prehistoric man seemed to have worn the teeth down very early, and so produced a flat surface, which was not favourable for the development of caries. There was no evidence, so far as he knew, of post-nasal growths.

Odontological Section.

November 23, 1908.

Mr. J. HOWARD MUMMERY, President of the Section, in the Chair.

Adenoids and the Feeding of Infants in Relation to the Growth of the Jaws.

By J. F. COLYER.

IN order to ascertain whether methods of feeding in any way influence the growth of the palate and dental arches, I have examined a series of models and patients. The material has been obtained from private and hospital practice, the number of cases investigated with a definite history of the feeding, &c., being over 300. A certain amount of difficulty has been experienced in obtaining material in hospital practice, owing to the fact that so many children have such mutilated arches that the models are valueless for measurements.

In dealing with cases one has to be careful to exclude the possibility of error in occlusion and measurements due to early extraction of the deciduous teeth. With private patients this is an easier task than with those coming under notice at the hospital. I feel, too, that the amount of material examined is not sufficient to draw any very definite conclusions. Still, even taking into account these disadvantages, the data so far obtained are interesting, and I have ventured to bring them to your notice with the hope that they may arouse your interest in the subject, for it is only by patient investigations that we can hope to advance our knowledge of the etiology of deformities of the dental arches and palate, and not until we have that knowledge will it be possible for treatment to be rational

MEASUREMENTS AND NOTATION.

For the purpose of obtaining measurements, the following points have been utilized:—

(a) The breadth between the first permanent molars, the point chosen for measurement being the gum margin immediately in line with the fissure in the palatine aspect of the tooth. This measurement is denoted throughout by A.

(b) The breadth between the first deciduous molars or first premolars, the point chosen being the gum margin at the point corresponding to the centre of the palatine aspect. This measurement is denoted throughout by B.

(c) For the deciduous teeth points between the second deciduous molars and canines were selected — denoted by the letters C and D respectively.

In selecting points near the gum margin rather than the cusps it seemed that a truer idea of the breadth of the palate could be obtained. The width between the cusps varies greatly with the alignment or tilt of the tooth and, still further, does not give the width of the palate. The following notation was adopted in recording the occlusion of the teeth:—

N to indicate that the occlusion of the teeth was the normal, namely, mesial surface of upper molars posterior to that of the lower (fig. 1).

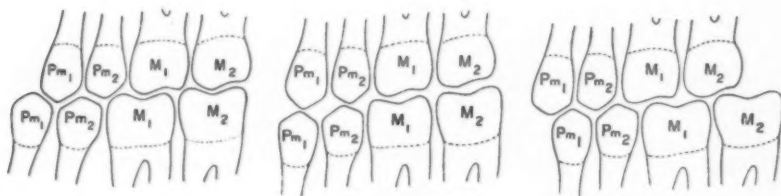


FIG. 1.

FIG. 2.

FIG. 3.

X to indicate that the mesial surface of the molars or premolars were flush (fig. 2).

O to indicate that in occlusion the mesial aspects of the maxillary teeth were in advance of the mandibular (fig. 3).

To obtain an idea of the shape of the palate plaster casts were obtained of 100 cases. These were divided at a point corresponding to the middle of the second deciduous molar or second premolar, and also through the median line. By means of these two sections a

fair idea of the shape of the arch of the palate was obtained. The measurements throughout are expressed in millimetres.

Of the cases in which adenoids were not and had not been present, and there was no history of such habits as thumb sucking and the use of comforters, only 38 were breast-fed. The term breast-fed was limited to those in which this method of feeding had been adopted for at least six months. Very few children in private practice were breast-fed for periods longer than this, but in the cases of hospital patients the period was generally from nine to twelve months, and more often the latter than the former.

The average measurement of the breast-fed cases was :—

		A		B
Private cases (20)	...	34.85 mm.	...	26.31 mm.
Hospital cases (18)	...	34.55 "	...	26.43 "

These figures show a slightly increased breadth in the molar region in private cases and a decreased breadth in the premolar region—in other words, the curve of the arch in the well-to-do child is greater than that met with in those not so favourably placed. Taking the average of both classes, the 38 cases show the following figures :—

A		B
34.70 mm.	...	26.36 mm.

the difference between the two measurements being 8.34 mm.

The occlusions work out as follows (in 3 of the hospital cases the occlusion could not be correctly ascertained) :—

Right side	Left side		Private cases		Hospital cases
N	N	...	5	...	8
N	X	...	2	...	4
X	X	...	2	...	2
O	O	...	—	...	1
N	O	...	1	...	—

Twenty-three, or 65.7 per cent., are normal, and in only 2 cases is there an O occlusion. Counting each case as two occlusions, we find out of a possible 70 there are 53 normals, or 75.7 per cent. Taking private cases only, the average is 75 per cent. normals on both sides, and 33 N out of a possible 40. It is not easy to see the cause of the O occlusions; in both cases the deciduous molars were still present, and I was not able to obtain a history of the use of the comforter or thumb sucking.

The O occlusion is at times foreshadowed in the deciduous dentition. In one example, for instance, the child was breast-fed for nine months, was perfectly free from adenoids, and had never used the comforter, &c.;

the width of the arch was slightly above the average, yet the occlusion was O X.

The numbers of breast-fed children aged under 6 examined have been small, namely, 13. The average measurement of these was:—

C	D
29.65 mm.	22.76 mm.

Turning to a consideration of hand-fed children free from adenoids, 34 cases in private practice yielded the following figures:—

A	B
34.1 mm.	25.61 mm.

the occlusion in 33 cases being:—

Right side	Left side	Private cases
N	N	21
N	X	6
X	O	2
X	X	4
N	O	—

The normal occlusions amount to 63.6 per cent. Taking the total possible occlusions we find 48 out of 66, or 72.7 per cent., normal.

A comparison of these figures with those of breast-fed children (private) shows:—

	A	B
Breast-fed	34.85 mm.	26.31 mm.
Hand-fed	34.1 "	25.61 "

These figures show that breast-fed children have slightly better-developed arches than those that are hand-fed. Amongst my hospital patients the number of hand-fed children without adenoids was too small for comparison with those breast-fed. The few figures I have appear to indicate that the difference is more marked than in the case of private patients. What is the cause of the variation in size between the breast- and hand-fed? The answer is probably to be found in the difference between the action of the teat and the nipple on the palate.

To quote Dr. Pedley: "If we watch an infant in the act of taking its natural food, we see that it opens its mouth to the fullest extent and takes a mouthful of its mother's breast; not only the nipple, but a large part of the areola disappears. At the same time the tongue is protruded over the whole of the lower gums and often over the lip, and its edge may be seen in the angles of the lips. Thus an infant aged 3 months takes into its mouth a soft, tough mass, which, when its jaws close upon it, measures from 1 in. to $1\frac{1}{2}$ in. wide, $\frac{1}{2}$ in. in thickness,

and 1 in. to $1\frac{1}{2}$ in. long—a flattened truncated cone, which, when subjected to the pressure of the tongue and the jaws, comes into contact with the larger portion of the palate and the upper gums, and the upper surface of the tongue. The nipple itself forms a small part of what an infant aged 1 month takes into its mouth. The everted lips lie around the base of this soft cone, with slight, if any, contraction.

“In the act of feeding at the breast the jaws hardly close. The lower jaw is raised to squeeze the pap against the upper gums and the palate; a wave of contraction passes along the intervening tongue from its tip to its base, in the latter part of which movement there is some retraction of the body of the organ, which in this closed cavity makes for suction; then its approximation to the palate pushes the milk into the pharynx to be swallowed. The milk which is contained in the distended ampullæ immediately above the nipple is squeezed out rather than drawn out by suction. The jaws then separate to admit more milk into the flaccid apex of the breast, for the process to be repeated; the first part of this effort of expression is aided by the lower gums biting inside the upper. The cheeks are passive, the whole act is peristaltic, the alternating pressure and relaxation exerted by the jaws and tongue being analogous to that which the milker imparts with his fingers to the teat of the cow. The muscular effort becomes stronger as the meal terminates and ducts empty of secretion.” The general effect of the act is to bring the muscular tissue about the jaws into activity, and the effect on the palate is of a spreading character.

With the modern-shaped bottle the child is held on the left arm and the bottle is kept at such an angle that the milk passes into the teat; the mandible then exerts a squeezing action and the milk, passing into the mouth, is swallowed. If the bottle is used correctly, there is but little sucking on the part of the child, the whole act simulating that of the child at the breast; but there is this difference: the teat is not so tough and resistant as the breast, and the pad in the mouth exerts less pressure on the anterior part of the palate; still further, the backward force of the breast upon the anterior part of the arch is absent.

It would therefore seem that teats for bottles should be made flat and broad at the base, and a little longer than at present and of a tougher character—in other words, as near an approach as possible to the nipple and adjoining part of the breast. With so many children being hand-fed, the question is one of importance, and I think that the ideal teat has yet to be designed. Of those in use the Soxhlet is fairly good. It seems, however, to be just a little too long, and the child has a tendency to get

the teat too far back in the mouth. Its action on the palate is good, and some of the best arches I have seen in hand-fed children have been those in which the Soxhlet teat has been used.

The modern feeding-bottle is, however, a great advance on the old tube feeding-bottle. In this the teat, about 1 in. to 1½ in. long, is of a narrow tube-like type. In taking the milk from this bottle the tongue is closed around the teat and the act of suction is brought into play. The mandible is not used to any extent and the whole action of the muscular tissue of the cheeks is towards the median line and not away from it as is the case with the child at the breast. The action tends to narrow rather than spread the palate, and measurements of models bear evidence of this. In my cases there were 41 patients where the tube bottle had been used, and the average measurements were:—

A		B
32.14 mm.	...	23.05 mm.

These figures, compared with those of hand-fed by ordinary boat bottles, show a marked diminution in width, especially in the premolar region.

To investigate the shape of the palate 45 cases were examined, 20 breast-fed and 25 hand-fed. Tracings were made of the transverse and longitudinal outlines, and in the case of the former an attempt made to find out the breadth compared to the height. For this purpose a line was drawn between the gingival edges, and from the centre of this base a line was drawn at right angles to ascertain the height. The comparative breadth to height was ascertained. In the case of the breast-fed cases the breadth averaged 2.63 times the height, while in the case of the 25 hand-fed the figures were 2.52—a balance in favour of the breast-fed.

An examination of the tracings obtained showed that great variations exist in the shape of the palate, and that the arch of the palate is often asymmetrical. A series of these tracings is shown in fig. 4 (p. 16).

With the tube bottle the palatal arch is altered, as will be seen from the tracings of the three cases (fig. 6). These patients had not suffered from adenoids and so may be taken as examples of the action of the tube feeding-bottle. One is struck by the height of the arch, the average breadth to the height in four cases being 1.86 compared with 2.52 and 2.63 in cases of hand- and breast-fed children. The drawing marked (a) is of a child of Irish parentage, and even here, although the arch is broader than the average, the relation of breadth to height is 2.26, or well below the average of hand- and breast-fed.

ADENOIDS.

In examining the effect of adenoids all cases showing protrusions, or "open bite," were excluded, the investigations being limited to cases showing simple crowding of the arches. By this means it seemed possible to obtain a more accurate idea of the effect of adenoids on the arch and palate. Eighty-two cases were examined (42 breast-fed, 40 hand-fed). The measurements of these were:—

	A	B	Difference
Breast-fed (42) ...	33.39 mm.	24.42 mm.	8.97 mm.
Hand-fed (40) ...	33.01 "	24.51 "	8.50 "

If we compare the figures of the cases occurring in private and hospital practice with the normals one or two interesting points are brought to light:—

BREAST-FED.

Private cases ...	{ Normal ...	34.85 mm.	26.31 mm.
	{ Adenoids ...	33.80 "	25.19 "
	Difference ...	1.05 "	1.12 "
Hospital cases ...	{ Normal ...	34.55 "	26.43 "
	{ Adenoids ...	33.16 "	24.02 "
	Difference ...	1.39 "	2.41 "

HAND-FED.

Private cases ...	{ Normal ...	34.10 "	25.61 "
	{ Adenoids ...	33.56 "	24.90 "
	Difference ...	0.54 "	0.71 "

The occlusions of the cases were:—

BREAST-FED.

		PRIVATE CASES		HOSPITAL CASES	
Right side	Left side	Normal	Adenoids	Normal	Adenoids
N	N	15	6	8	6
N	X	2	—	4	2
X	X	2	4	2	8
O	O	—	—	1	3
N	O	1	—	—	1
X	O	—	3	—	1

HAND-FED.

N	N	21	16	—	3
N	X	6	4	—	2
X	X	4	3	—	2
O	O	—	1	—	—
N	O	—	—	—	—
X	O	2	6	—	1

or—	Breast-fed, private cases ...	{ Normal ...	75.0 per cent.
		{ Adenoids ...	46.1 "
	Breast-fed, hospital cases ...	{ Normal ...	53.3 "
		{ Adenoids ...	28.5 "
	Hand-fed, private cases ...	{ Normal ...	63.6 "
		{ Adenoids ...	53.3 "

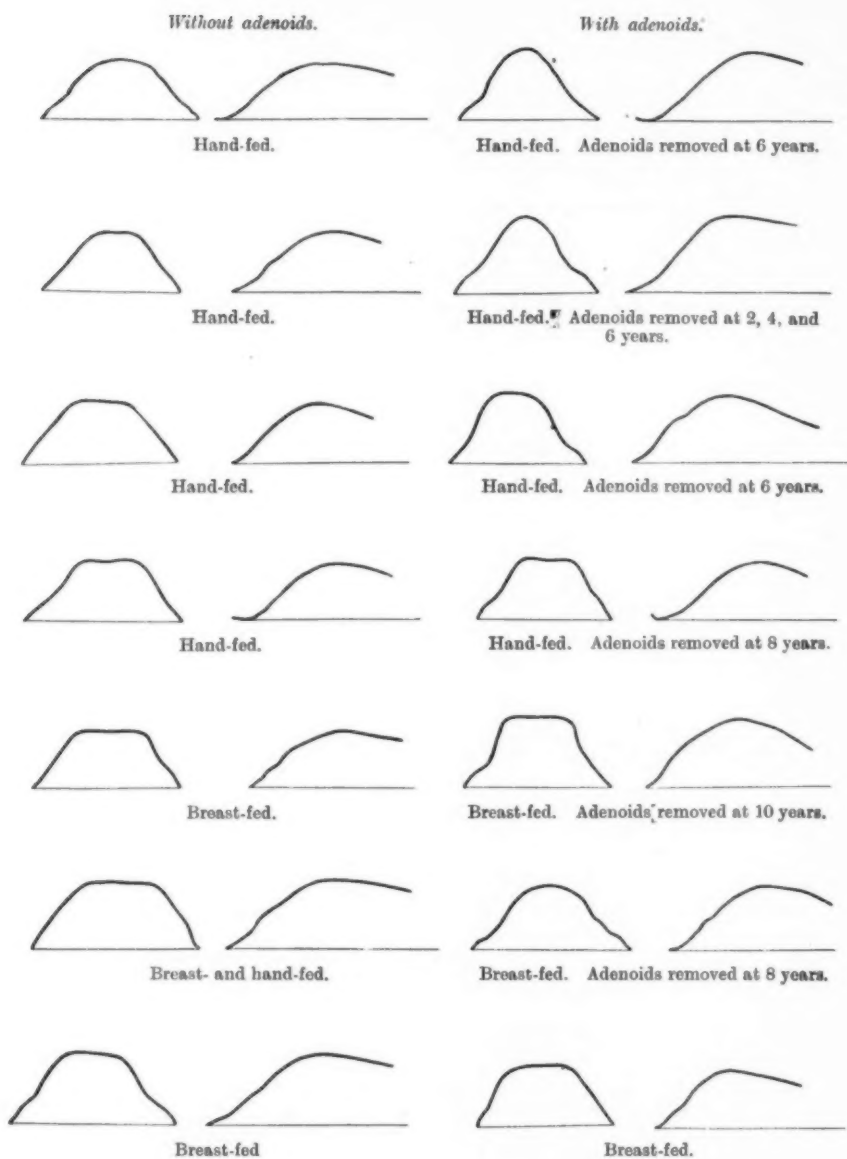
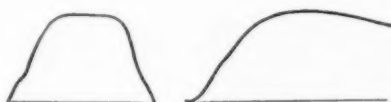


FIG. 4.

FIG. 5.

Tube bottles, without adenoids.



(a)

FIG. 6.

Tube bottles, with adenoids.



Adenoids removed at 5½ years.



Adenoids removed at 13 years. Came on before age of 6 years.



Adenoids removed at 15 years. Came on after age of 6 years.

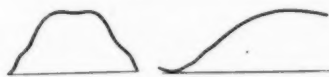
FIG. 7.

Protrusion associated with comforter but without adenoids.



FIG. 8.

Protrusion associated with comforter and adenoids.



Adenoids removed at 4½ years.



Adenoids removed at 8 years.



Adenoids removed at 7 years.

FIG. 9.

Unfortunately the number of hand-fed hospital patients without adenoids is too small to draw any inference from. The figures, however, suggest the following ideas:—

(1) That as far as breast-fed children are concerned the effect of adenoids is more marked in hospital than in private patients. This point, taken with the fact that adenoids go longer untreated in hospital than in private patients, suggests that some of the narrowing in adenoid palates is due to the action of the tissues of the cheeks.

(2) The change is more marked in the front than the back part of the mouth, again suggesting the pressure inwards of the cheeks. This would naturally be felt more in front than at the back of the arch.

(3) The effect of adenoids in narrowing the arch is less felt in hand-fed than breast-fed children. This point is of interest, and certainly requires further investigation.

The degree of deformity to the palate from adenoids depends mainly on the age of onset of the trouble and the period of continuance of the obstruction.

In the adenoid palate the ratio of breadth to height is less than in normals.

Breast-fed + adenoids (9 cases)	2.13
Hand-fed + adenoids (22 cases)	2.25

The normal figures are 2.63 and 2.52 respectively.

The alteration is greater in the breast-fed than in the hand-fed. This corresponds to the general changes in width and occlusion already noted. With regard to these figures, hospital and private cases are included. The majority of cases under "breast-fed" are hospital patients and under "hand-fed" are private patients.

Drawings of breast- and hand-fed children with adenoids are shown in figs. 5 and 7. It will be noticed that the arch of the palate is more irregular and that the rise of the palate from the incisor teeth is more abrupt than is the case in normals, the general idea given being that there has been some degree of lateral pressure brought on the sides of the arch, forcing it up.

If a series of models of children be examined in which adenoids were present in early years, the constant presence of more or less crowding of the incisors will be apparent. This crowding may involve all four incisors or only the lateral incisors, expressing itself as a slight tilt of these teeth. On examining such teeth more critically it will be noticed that the general trend of the roots is towards the median line. The apices

are squeezed, as it were, together. This crowding must be attributed to want of growth of the premaxillæ owing principally to lack of function of the anterior nares. I am inclined to think that the view of Mr. Tomes that the growth ceases between the premaxillæ and maxillæ at birth is open to question.

An examination of the skulls in the museum suggests that growth does continue to a much later date than is usually stated, and that the spacing of the incisors just previous to the eruption of the permanent teeth is in a certain measure connected with this growth. In several of the maxillæ showing well-marked spacing of the incisors the suture was well marked (fig. 10) while in others with the suture almost obliterated the spacing was absent. These associated conditions were, however, not constant, so that one is unable to draw any definite



FIG. 10.

conclusion. Some specimens showing marked spacing between all four teeth were associated with premaxillæ developed from two centres, and in one instance of spacing between the central incisors there is a suggestion of an extra suture running to the median side of the tooth. In one specimen the suture is carried back into the palate (see fig. 11).

If, then, as seems probable, growth does continue at the premaxillary-maxillary suture, the spacing of incisors seen in normal children is more rationally accounted for than by assuming a forward translation of the teeth by the tongue. Again, the crowding seen in adenoid cases can be explained on the lines of want of growth. This point is of the utmost importance with regard to the question of treatment.

A further effect of adenoids on the arch is to be seen in the region of the first molars. If, as seems probable, the growth of the maxilla and the eruption of the molar teeth are mainly dependent upon the growth of the antrum, in nasal obstruction the function of the antrum is

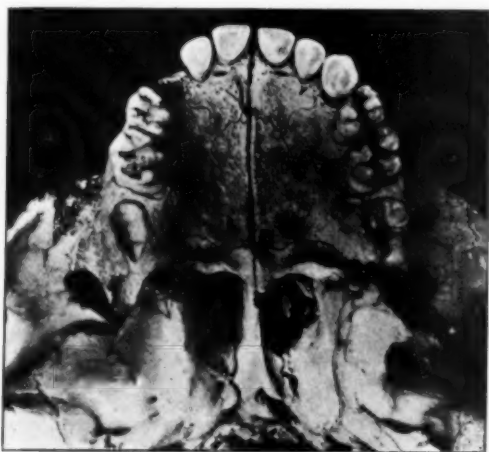


FIG. 11.

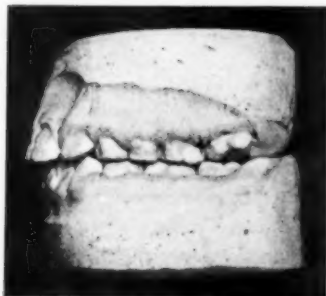


FIG. 12.

in abeyance, with the result that the orderly growth of the jaw is interfered with. The result of this is principally seen in a want of growth in the molar region, with the result that the molar teeth are crowded. The first molar when erupted often fails to assume a vertical position and lies with a general slope backwards. This is well seen in fig. 12,

the model of a child that suffered from adenoids. This tooth is in normal occlusion, but it is not difficult to see that directly the second deciduous molar is removed the pressure of the developing teeth will cause a rapid forward movement, and an abnormal occlusion will result, with an encroachment on the space for the premolars.

In some cases the want of growth in the molar regions is so marked that the first molar erupts in such a way as to lead to absorption of the posterior aspect of the second deciduous molar (*see* figs. 13 and 14).



FIG. 13.

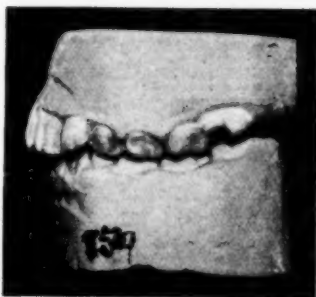


FIG. 14.

Under such conditions it is quite clear that abnormal occlusion must result, and the space for the premolars be considerably curtailed. A study, then, of the palates and arches of children with adenoids, in comparison with those of normals, points pretty definitely to these facts:—

(1) That there is a definite lack of growth of the bone, especially in the incisor and molar regions.

(2) That the pressure or weight of the cheeks tends to narrow the arch, the effect being more felt from this cause in the anterior than the posterior part.

SUPERIOR PROTRUSION.

The type of irregularity known as superior protrusion was seen in 159 cases—69 hospital, 90 private. Of these a complete history as to feeding and adenoids was obtained in 56 private and 29 hospital cases. The measurements were as follows :—

		Without adenoids			With adenoids	
		A	B		A	B
Private cases	...	34.03 mm.	24.53 mm.	...	32.53 mm.	24.03 mm.
Hospital cases	...	32.14 "	23.61 "	...	31.61 "	22.17 "

Out of 56 patients (private) whose history was ascertained, 8 only were breast-fed, and of those from hospital records 16. One has therefore insufficient data of breast-fed to draw any comparisons with cases not showing protrusion. The number of hand-fed cases is perhaps, however, sufficient for the purpose. The figures of the hand-fed private cases are :—

Private cases		A		B	
Normal, without adenoids	...	34.10 mm.	...	25.61 mm.	...
Protrusion without adenoids	...	33.95 "	...	24.55 "	...
Ordinary cases with adenoids	...	33.56 "	...	24.90 "	...
Protrusion with adenoids	...	32.48 "	...	24.31 "	...

These figures show in the case of non-adenoid children an important narrowing in the premolar region in the case of protrusion; in the case of adenoids a slight reduction in the premolar region, with a larger one in the molar. In the case of 14 hospital cases of protrusion plus adenoids, the figures are as low as :—

A	B
31.32 mm.	21.32 mm.

The most interesting facts come to light in studying the question of occlusion. They are :—

Right side	Left side	PRIVATE CASES		HOSPITAL CASES	
		— Adenoids	+ Adenoids	— Adenoids	+ Adenoids
N	N	...	2	...	7
N	X	...	1	...	4
X	O	...	—	...	4
X	X	...	5	...	4
O	O	...	5	...	1
N	O	...	—	...	2

Out of the 72 cases there were only 15 normal, or 20.83 per cent., while we find 24, or just 33.3 per cent., with an O occlusion of the teeth

on both sides. If these figures are compared with those of adenoid cases, unaccompanied by protrusion, the striking feature is not so much the decrease in normal occlusion but the great percentage of O occlusions. In 140 occlusions (breast- and hand-fed, private and hospital) without adenoids, there were only 5 cases of "too forward occlusions" on both sides.

This abnormal forward occlusion means either a normal maxilla with a defectively developed mandible or a movement forward of the whole maxillary arch. There are many who maintain that protrusion of the upper teeth is the result of the former condition, but I have never yet seen any satisfactory data brought forward to support the statement, and they have never pointed out whether the want of growth of the mandible is due to congenital defect or due to disuse through want of function. If the former, then the difficulty must be explained why the mandible should be the only bone in the body so liable to congenital defect, especially when we consider the relative scarcity of such defects in other bones. If the latter contention, namely, that the defect is due to disuse from lack of function, then another difficulty has to be met and explained. Tissues, and therefore bones, depend for development upon the "inherent power of growth" and functional activity. With regard to "inherent power of growth," there is no reason why this should be interfered with more in the mandible than in the maxilla, and in connexion with the question of functional activity it must be remembered that the mandible is a freely movable bone, whose main function is concerned in the process of mastication, whereas in the maxilla (a fixed bone) growth is affected not only by the proper carrying on of the process of mastication, but also by efficient nasal respiration. Now, although we know that the modern-day food has reduced the function of mastication, it is fair to infer that the growth of both maxilla and mandible has been affected; but this is common to the race, and there is no reason to believe that the influence is more marked in the mandible than the maxilla. On the other hand, the frequent presence of adenoids, and the fact that lack of nasal respiration interferes in a marked way with the growth of the maxilla, points to the maxilla being the bone under-developed.

In fig. 15 is shown a case of superior protrusion due to a forward movement of the maxillary teeth.

I do not contend that the mandible is never at fault in superior protrusion. I think it is in a few cases, but it is rather to the maxilla we must turn for an explanation of protruding teeth.

If a series of children be examined with adenoids the larger section will be found to have simply a crowded condition of the teeth and the smaller section superior protrusion. Adenoids, we have seen, narrow the arch and generally interfere with growth of the bones, but why in one case does a simple crowded condition of the teeth result, and in another a general protrusion frequently unaccompanied by much displacement from a regular arch, and in other examples the deformity known as "open bite"? To take first the private cases breast-fed without adenoids, the following facts were obtained:—

Occlusion		Measurements in millimetres		Feeding	Comforter and thumbs
Right side	Left side				
		A	B		
—	—	—	—	Breast- and hand-fed	Comforter eighteen months
O	O	33.5	24	Breast- and hand-fed	Thumbs constantly
N	N	32	24	Hand-fed	Thumbs three years
X	X	32	25	Breast- and hand-fed	Lip constantly
O	O	35	27	Hand-fed	Comforter two years, rickets
O	O	29.5	20.5	Breast- and hand-fed	Thumbs
X	X	34.5	24.5	Breast-fed	Thumbs until aged 5
N	N	33	26	Breast-fed	Comforter fifteen months
X	X	36	27	Breast- and hand-fed	Frænum
X	N	35	26	Hand-fed	Comforter about fifteen months
X	X	37	24	Hand-fed	Thumbs
X	X	33	21	Breast- and hand-fed	Fingers one year, tube bottle
O	O	35.5	23	Breast-fed	Thumbs three years
O	O	36.5	27	Hand-fed	Comforter two years
		C	D		
(15) O	O	27	20	Hand-fed	Comforter three months, rickets
N	N	29	22.5	Breast-fed	Comforter fifteen months
X	X	31.5	24	Hand-fed	Thumbs until aged 5
N	X	30	20	Hand-fed	Comforter a long time

In 16 cases out of the 18 there was a history of the use of the comforter or thumb sucking, in one case the protrusion was definitely due to sucking the lower lip, and in one the only ascertainable abnormality was the frænum. In the case numbered 15 I had the opportunity of seeing this child when aged about 18 months, and the protrusion was then well marked (fig. 16).

Of the cases accompanied by adenoids there was a history of the use of the comforter or thumbs in 19 of the children for periods in some cases up to the age of 3. In 5 of these the tube bottle had been used. Of the remaining cases, in 11 there was no history of the comforter or thumbs, but in three of these the tube bottle had been used. In 8 cases the history of comforter, thumbs, &c., was not inquired about.

Of 9 hospital cases without adenoids, in 6 there was a history of the comforter, thumbs, or tube bottle; while of 24 cases with adenoids, in 18 there was a history of some deforming force, in 1 the deciduous teeth had been knocked out when aged 3, and of the remaining 5, 2 showed hypoplastic teeth, and 1 definite signs of previous rickets.



FIG. 15.

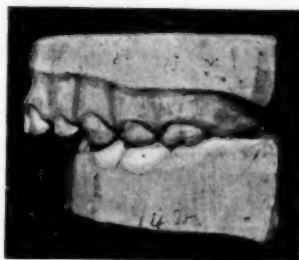


FIG. 16.

These figures are strongly suggestive of the comforter, fingers, or tube bottle as being the determining factors in the production of

D—20

protruding upper teeth. In a few cases there is only a history of adenoids, but how this condition produces adenoids is not clear.

I admit that a history of tube bottles, comforters, and thumbs is met with in those not presenting protruding teeth, and although my statistics are not very extensive on this point, I find that in 163 private patients in whom I have inquired as to the use of a comforter, &c., 107 had not used it, and of these 13 showed protrusion. A history of the use of comforter or thumbs was ascertained in 56 cases, and of these 39 showed definite protrusions. The slight use of a comforter for the purpose, perhaps, of sending the child to sleep probably does no damage; it is rather the persistent use over a long period.

It is quite probable that it will be argued that the comforter is not so fruitful a source of trouble as my figures seem to suggest, because protrusions do not, as a rule, arise until the eruption of the second dentition. My observations lead me to believe that this is not the case, and that slight protrusion of the deciduous incisors is very common in the first set, and is the forerunner of a more apparent deformity in later childhood. A slight protrusion in early years may easily go unnoticed, and this, combined with the small number of children under the age of 6 brought to the dental surgeon, may account for their supposed absence.

My attention was first drawn to the question of the use of the comforter by a paper contributed to the *British Medical Journal*, 1906, ii, p. 989, by Dr. T. Pedley, but I little expected to find on inquiring into the subject how his views would be confirmed by one's own observations. I think myself there can be no doubt that the comforter and thumb sucking must be regarded as a most important factor in the production of protruding teeth.

The production of protruding teeth in cases associated with adenoids alone is not easily explained, and I do not propose to-night to discuss the matter. The majority of such cases have the lower incisors driving on to the upper teeth, and are associated with want of growth in the molar region. Sections of palates of protrusion associated with comforter but without adenoids are shown in fig. 8 and of protrusions with comforter and adenoids in fig. 9.

M. H. Cryer, in an excellent paper on "Factors that Modify the Human Jaws and Face,"¹ clearly recognizes the evil results that may follow prolonged thumb sucking or the use of a comforter; he says: "Mouth breathing and sucking of the thumb or any foreign substance

¹ *Dental Cosmos*, 1906, xlviii, p. 1082.

while the mouth is undergoing development will modify, not only the shape of the mouth itself, but that of the whole face. When the jaws are apart the action of the various muscles tends to draw them inward. The hyoid group has a tendency to draw the mandible in at the angles. The palato-glossus and other muscles of the region also draw the tuberosity of the maxilla inward and the facial muscles are presumed to act in a similar manner. The sucking of the thumb or of false nipples puts into action the muscles connected with the orbicularis oris, which action, in concert with that of the palato-glossus and the palato-pharyngeal, assists in bringing about a contraction of the arches."

"OPEN BITE."

In referring to "open bite" I propose to limit my remarks to that variety in which the lack of occlusion involves the premolar and molar region. Thirty cases were examined. In 24 of these, measurements were taken with the following results:—

A		B
30.56 mm.	...	21.35 mm.

figures which illustrate a considerable narrowing of the arch, especially in the molar region. The records of the occlusions in 21 of these are:—

Right side	Left side		
N	N	...	6
N	X	...	5
X	X	...	4
X	O	...	4
O	O	...	3

If these are compared with the occlusions of superior protrusion it will be noticed that the percentage of normal occlusions is higher and of too forward occlusions far less in cases of "open bite."

In 25 of the 30 cases hypoplastic teeth were present, and this is a point of some importance. Hypoplastic teeth may be regarded as a sign of previous rickets, but even if this is open to question they indicate that the blood-stream to the jaws was deficient in inorganic salts, and if the teeth suffered there is no reason why the nutrition of the bone should not also have been interfered with. This close association of rickets with "open bite" throws considerable light on the production of the deformity.

In 16 of the cases the history as to feeding and the condition of the naso-pharynx was obtained. The details of these cases are as follows:—

Feeding	Adenoids	Comforter	Thumbs	Hypoplastic teeth
Hand-fed	+	Constantly	—	+
Breast-fed	+	—	—	+
Hand-fed	+	—	—	+
Breast-fed, followed by Mellin's	+	Nineteen months	—	+
Hand-fed	+	Twelve months	—	+
Breast-fed	—	Three months	+	+
Breast-fed, long illness, rusks, biscuits	+	—	—	—
Hand-fed (Nestlé)	+	—	—	+
Breast-fed two years, Robb's biscuits after nine months	+	—	—	+
Hand-fed	+	—	—	+
Breast-fed (Neave's food) ...	+	—	—	+
Hand-fed	+	—	—	+
Breast-fed	+	—	—	+
Breast- and hand-fed	+	—	—	+
Breast-fed	+	Nineteen months	—	+
Breast-fed	+	—	—	+

The points to notice are (1) in all cases except one adenoids were present; (2) in 15 cases hypoplastic teeth were present; (3) in 12 of the cases in which inquiry was made about the comforter, 5 had used it.

These facts seem to support the view I advanced in a previous paper,¹ namely, that "open bite" is due to the ill-effects of adenoids plus rickets. We know that the weight of the cheeks tends to narrow the jaw; if, now, this weight is acting on easily pliable bones the effect will be accentuated. In severe cases the mandible takes a marked bend down about the anterior border of the insertion of the masseter; this condition is probably due to a bending of the bone, due to the downward pull of the muscles opening the mandible counteracted by the elevators of the mandible. I am also inclined to think that the upward displacement of the maxillary incisors, which is at times present, is due to the action of the comforter or the teat of the bottle on bones softened by rickets. In other words, the agencies which produce general crowding and protrusion, when combined with rickets, produce grosser lesions, which show themselves in the form of "open bite."

¹ *Trans. Odont. Soc., Lond., 1896, xxix, p. 31.*

DISCUSSION.

The PRESIDENT (Mr. Howard Mummary) said that a very large field was opened up by the paper, and he hoped it would meet with a good discussion. Mr. Colyer's observations with regard to the different actions of the jaws of the child produced by the two different forms of feeding-bottle were of decidedly great interest. He thought that most pernicious invention the "comforter" was responsible for a great deal of evil and should be condemned on more grounds than one.

Dr. SIM WALLACE said there were many points in the paper with which he agreed, but he did not intend to refer to them, desiring only to put forward some interpretations of the author's figures that he thought were perhaps as good as the interpretation given by the author himself. With regard to the measurements of breast-fed children in private and in hospital patients, although the difference was not great there was a difference in the measurement between the private and hospital patient, although they had been fed in exactly the same way; to a certain extent the hospital patients even fared better, inasmuch as the breast-feeding had been continued rather longer, and possibly that might account for the very slight increase in the premolar region. In the molar region, the region where a tooth came into its position at a later time, when other things had had an opportunity to take effect, there was a definite, although a small difference in the private patient and the hospital patient. The difference no doubt resulted from the fact that hospital patients did not live in such hygienic surroundings as more favoured children and consequently were not so well nourished. Taking statistics of the weights of children brought up in the wealthy classes and those brought up in the poorer classes, there was a distinctly greater body-weight in the well-to-do children; in general their muscular development was greater, and amongst the muscles was that important organ the tongue. With regard to bottle-fed children, it appeared from what the author had said that the greater difference was in the hospital patients; those that had been fed less hygienically were rather less well nourished than the children of the well-to-do, who had more fortunate hygienic surroundings. The same fact was observable in a comparison of the Soxhlet bottle with the tube bottle. The tube bottle had been given up on account of the difficulty of sterilizing the tubes, whereas the Soxhlet bottle had no tube, the top being readily taken off and the rubber teat turned inside out and cleaned and sterilized. Therefore he thought the differences the author had pointed out in his statistics were explicable on another theory altogether; there might be some slight difference in the effect of the comforter compared with the breast, but the measurements were well explained by the fact that the tongue was an important organ in the moulding of the dental arches. In the case of adenoids associated with mouth-breathing the function of the tongue was in

abeyance in the upper jaw and there was consequently an arrest of development in the maxilla. He agreed with the author in attributing superior protrusion largely to the lack of development of the maxilla. One slide showed very well how the narrowing took place; teeth were trying to come into position where there was insufficient space for them, the bony growth which should have taken place on the posterior part of the maxilla had not taken place, and consequently the teeth came crowding into a position where there was no bony substance for them; whereas in a well-developed jaw the first molar came into position not in contact with the second temporary molar, but actually with a tiny space between. If the first molar came into position and crowded all the teeth forward it obliterated the spaces and tipped all the teeth forward. The author seemed to think that the tongue pressing on the arch was not a very good explanation of why the spacing of the teeth came into existence, but as a matter of fact the spacing not only came into existence in the intermaxillary-maxillary suture, but between the incisors also in the upper jaw, as well as between all the front teeth in the lower jaw where there was no intermaxillary-maxillary suture. In the case of adenoids the tongue did not carry the mandible and the maxilla forward, and consequently the developmental stimulus that should be given to the deposit of bone by the transference of the maxilla bodily forward was not given, and crowding occurred. If the tongue was admitted to be the principal stimulus in carrying the jaws forward and stimulating the growth posteriorly, the gradual and normal growth could be accounted for. Although he admitted that the comforter, or the thumb, or any other thing kept continuously in the mouth might produce irregularities, yet as the tongue was the most constantly present, so it was of the utmost importance to see that it was normal in size, position, and relation to the jaws, especially if it was desired to prevent irregularities.

Mr. F. J. BENNETT said the question turned upon the effect of the comforter and breast-feeding, and in dealing with such a question the proper time to make an observation was at the end of the period of breast- or hand-feeding, taking a model of the jaw at the end of the ninth or twelfth month. That would set at rest innumerable questions which might be speculative when the models were taken at the age of 5 or 6 years. A multitude of things had happened to the child during that time. In looking at the lips of a child during the period of suckling, up to the ninth month, one found a characteristic shape indicative of the muscular action of sucking, but the lips became totally different in shape by the time the child was 4, 5, or 6 years old. If the change in the lips was the result of a different method of feeding, it might also happen that a change also occurred between the time of suckling being given up and the fourth or fifth year. The author appeared to assume that all the conformation of the jaw took place during the period of suckling, but surely a great number of changes took place afterwards? It was so with the lips, and why not with the palate? The author suggested that the intermaxillary sutures might be open at a later period than was usually imagined, but that was a thing that could be settled in ten minutes by the use of the X-rays. Speculation

on the subject was unnecessary. He thought a great many things that were put down to the use of the comforter and the difference between hand-feeding and breast-feeding had been overlooked. One was the general condition of the parents. The fact that children were brought up by the bottle betokened to some extent an enfeebled condition of the mother, and it might be due to that enfeebled condition rather than to the fact that the child was hand-fed. Taking half a dozen children brought up at the breast there would be a considerable variation in stature, weight, and other matters, and it might also be that the jaws might vary in the same way. In those respects he thought there were some fallacies which the author might have omitted, and that he might have had more success if he had taken models at an earlier period in the child's life. But in saying those things he did not wish to disparage the value of the paper, recognizing it as a valuable contribution.

Mr. W. H. DOLAMORE, while recognizing the extraordinary amount of work the author had devoted to the subject, could not believe that the deformities were wholly due to a mechanical action in bottle-feeding, or the use of the comforter, or to adenoids. It seemed to him that the amount of distortion which would occur in bottle-feeding, or by the use of a comforter, in the small mouth of a little child—the bottle-feeding going on for one year and the comforter generally being dropped before the second year—would be very slight, and, indeed, the author's figures showed it to be so, because the difference shown in the tables in the paper between a hand-fed child and a breast-fed child was something under a millimetre, and, as a millimetre was something under $\frac{1}{8}$ in., this was not a very great difference. It seemed to him that, as Dr. Wallace had hinted, the cause was a deficient growth of the bone; that, if there was a difference between a breast-fed and hand-fed child, it was more dependent on the growth of the jaws afterwards than on the effects of mechanical action in early days. He had two models, the first that of a child now 14 years of age, who had adenoids which were not removed until she was aged 10. She was bottle-fed and used the comforter until her brother was old enough to know that he ought to throw his out of the window and she threw hers out too. He was a year younger than she was. He thought Mr. Colyer would regard the model as that of a very good type of jaw. The other model was that of a sister and showed a much greater difference than the author had been able to show between hand-fed and breast-fed children. The sister was also hand-fed and had adenoids, but they were removed when she was 4 years of age. In her case the measurement between the two first permanent molars taken at the gum margin was 5 mm. less than in the elder sister, yet the four incisor teeth measured 4 mm. more than the teeth of the elder child. Consequently the larger teeth of the younger child would have to crowd into a smaller arch than was the case with the older child. Granting that it might be all due to bottle-feeding, to the use of the comforter, or to adenoids, it was apparent that what the younger child wanted was a greater growth of jaw rather than any movement

of teeth themselves. Although the author had not entered into the question of the action of adenoids in a mechanical way producing irregularities, the same thing held good, and the so-called mechanical explanation, pressure of lips and so on, was very incomplete and unsatisfactory. There was one diagram in the paper of a child whose adenoids were not removed until the age of 15, and the author pointed out that the longer the adenoids were left the worse was their action. The diagram, however, showed a remarkably good arch, and compared very favourably with that of some of the cases in which the adenoids were removed quite early. He should also like to ask the author to explain how the growth of the antrum affected the growth of the jaw. He could quite understand the two growing coincidently, but how the growth of a cavity could assist the growth of the bone he could not understand. The author suggested, more or less, that the blocking of the ostium might diminish the growth of the antrum. It seemed to him that air in blowing through the nose would, if anything, produce a lower pressure in the antrum rather than an increased pressure, on the same principle that was employed in the "saliva ejector." If air did pass into the antrum when the nose was blown, it was obvious that whenever there was anything septic in the nose it must be blown into the antrum and would lead to a very severe inflammation if not to empyema. He could not help thinking that the growth of the jaw was prior to the growth of the antrum. It was well known that in drilling into antra the thickness of the bone which had to be drilled through varied immensely, and in some cases no cavity could be discovered at all. Therefore, it might be assumed, where there was no cavity there ought to be no jaw, according to the author's view; at any rate he would be glad of further explanations.

Mr. A. H. JOHNSON asked Mr. Dolamore if either or both of his cases suffered from rickets.

Mr. BREESE pointed out that there was a lack of dental irregularity and deformity amongst the Jewish race. He had been working at a large Jewish orphanage at Norwood for the past two years, and had found that serious cases of irregularity were almost non-existent. It might be said that that was a racial characteristic, but it was rather significant that the Jewish mother invariably nursed her own child—almost without exception the children were breast-fed. The question was gone into five years ago by Dr. William Hall, of Leeds, who examined 500 children of Jewish parents, and also the same number of Gentile children, and although his results were rather roughly grouped together, he stated that the Jewish child with a high and deformed palate was a rarity, whilst such children were very common amongst the Gentiles.

Mr. GEORGE NORTHCROFT congratulated the author very heartily on his paper, and wished other members would put the same amount of energy into similar work, because thereby the labours of the profession would be considerably lightened. There were one or two things in the paper that appeared to him to traverse the author's general conclusions. In the summary of the

conclusions on breast-feeding the author stated there were two O occlusions which he could not account for, and in the superior protrusions without adenoids there were five O cases. It seemed to him that such irregularities of occlusion must be accounted for in some other way than bottle-feeding, adenoids, thumb-sucking, or the comforter, and that these things could be only aiding rather than predisposing causes; he thought they could not be the origin of the condition. He was very glad to hear definite mention of the study of occlusion, because it seemed to him to be a thing that in the past had been sadly neglected. It did not of course matter to Mr. J. G. Turner, who considered that occlusion was of no value at all. There was one type of occlusion which the O and the N and the X did not include, what might be called the Z, the angle class, three cases. He thought it was a little unfair for the author to give the value of OO 2, and OX 1, because he doubled the value when the malocclusion was bilateral. An unilateral case was quite as difficult to treat as a double case, and it seemed to him it was an unfair statistical conclusion to state that the bilateral had really the value of two as compared with a unilateral case.

Alveolar Abscess in the Tooth of a Dog.

By GEORGE THOMSON, L.D.S.

THIS is the skull of a small retriever which Mr. Sewell, the eminent veterinary surgeon, brought to me. He told me that he had been treating it for six months for a sinus near the left eye. The skull shows what has happened quite obviously. It is discoloured from the left canine as far as the junction of the frontal bone, extending across the nasal processes to the maxilla of the opposite side and along the margin of the left orbit. The dog died after six months. The point of interest is the remoteness of the sinus from the cause of the discharge. Veterinary surgeons often meet with a fistulous opening below the inner canthus of the eye and are in the habit of extracting the fourth premolar for its treatment. Hobday's book on "The Dog" refers to this as being usually a fistulous opening into the antrum and recommends the extraction of the fourth premolar, and says that the tooth should be extracted and the fangs if possible. On reflection I remember that I had a patient with an opening in the cheek for twenty-four years before a diagnosis was reached. Several times he had had teeth extracted without the cause being found. I also remember that a patient of mine,

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a Fellow of the Royal College of Surgeons, had been going about for ten years with a fistulous opening in the cheek and the cause had not been discovered. Another point of interest is that the canine tooth is not often found by veterinary surgeons to be affected with alveolar abscess. The diagnosis and treatment of this would have been simple to a dental surgeon because there is a minute exposure of the pulp. The canine is the most difficult tooth to extract in a dog on account of its curves.

Odontological Section.

January 25, 1909.

Mr. J. HOWARD MUMMERY, President of the Section, in the Chair.

The Treatment of a Denture, Swallowed and Impacted in the Œsophagus, by means of Killian's Œsophageal Tube.

By PHILIP TURNER, M.S.

IN September last a patient was admitted into Guy's Hospital having swallowed a denture which had become impacted in the œsophagus. I will first give a brief account of the case and afterwards discuss the treatment.

The patient, a woman aged 53, was seen about six hours after the accident, which had occurred about 6 a.m. The plate was of irregular shape, its greatest length being $1\frac{1}{4}$ in., and breadth 1 in. It supported a single incisor tooth, and from it projected two hooks. Since the accident no solid food had been taken, though she could swallow fluids with a certain amount of pain. With the X-rays the plate could be distinctly seen behind the upper border of the manubrium sterni. Chloroform was administered, and when the patient was anæsthetized the head was fully extended over a sand pillow. A Killian's direct-vision œsophageal tube was then introduced, and when it had passed about 9 in. beyond the teeth the denture came into view and I was able to show it to those present in the operating-room. A long hook was passed through the tube, and by a little manipulation the denture was freed from the œsophageal wall; an attempt was next made to draw it upwards, but, as expected, it was too large to pass along the lumen of the tube. The plate was then, by means of the hook, drawn against the end of the tube, and when firmly gripped an attempt was made to withdraw the tube, hook, and denture together. As they passed through the pharynx the denture was felt to escape from the grip and drop, presumably against the posterior pharyngeal wall. The patient had now come round from the anæsthetic to such an extent that swallowing

and coughing reflexes were present. As a hurried examination of the pharynx and naso-pharynx did not reveal the presence of the denture more anæsthetic was given and the œsophagus again examined by means of the tube. This could, however, now be passed to the lower end of the œsophagus without seeing the denture, though the site of the former impaction was revealed by some bleeding at this situation. Presumably the denture fell to the back of the pharynx, and owing to the reflex swallowing movements it was projected along the œsophagus, this time without becoming impacted.

The next day skiagraphic examination showed the plate slightly above and to the left of the umbilicus, presumably in the stomach. The following day it was in the right iliac fossa, in the cæcum, and the next day in the transverse colon, while on the fourth day it was passed *per anum*, having given no trouble in its passage through the alimentary canal.

In this case, though the denture was too large to be withdrawn through the tube, yet by means of the latter one was able to see it *in situ* and to free it easily and safely from the wall of the œsophagus. When once in the stomach one felt certain that a foreign body of this size would safely traverse the intestinal canal.

Last month I was called upon to treat a child (E. C., aged 11) who had swallowed a pin which had stuck just at the commencement of the œsophagus. That it was impacted was shown by the fact that vomiting during the induction of anæsthesia did not dislodge the pin. In this case the pin was easily seen through a short tube and was then seized by forceps and withdrawn, the child leaving the hospital quite well on the following day.

The commonest foreign bodies to become impacted in the œsophagus are coins. The patients are usually small children, and, as a rule, the foreign body may be easily extracted by means of the coin-catcher, or displaced and pushed down into the stomach by means of a probang. Impaction of a tooth-plate, or of a pointed object such as a pin, is a much more serious accident, for the irregularities are likely to be caught in the œsophageal wall, and unless the foreign body is quickly removed ulceration, and perforation of the œsophagus are likely to occur, when a fatal suppuration extending into the mediastinum will result. Hence these cases should always be treated at once.

The use of the coin-catcher or probang in these cases is attended with a very great risk, for they are very liable to cause laceration of the œsophageal wall and thus to cause a suppurative mediastinitis.

Impaction of a denture in the œsophagus for these reasons must be

regarded as a very serious accident. A foreign body in the œsophagus is likely to become impacted in one of the three following places: (a) its commencement behind the cricoid cartilage; (b) where it is crossed by the left bronchus; (c) at the cardiac orifice of the stomach. In the first two situations the usual method of treatment, after failure to displace the foreign body by forceps or probang, has been to open the œsophagus by an incision in the neck and to extract the denture by means of forceps introduced through the wound. In the latter situation the stomach may be opened and the foreign body withdrawn by forceps introduced from below, through the cardiac orifice. The operation of incising the œsophagus or œsophagotomy is very serious, since leakage is likely to result, setting up fatal suppuration.

In the past eighteen years six cases of dentures impacted in the œsophagus have been admitted to Guy's Hospital, excluding the one I have described. Of these one was displaced downwards by means of a probang, afterwards passing *per anum*. Four were removed by œsophagotomy, of which one recovered and three died, two of suppurative mediastinitis and one of cardiac failure. One case was treated by gastrotomy, but here the plate had been impacted at the lower end of the œsophagus for seven weeks and could not be removed, the patient dying of shock shortly after the operation. In addition to these cases of impacted denture, however, Sir Alfred Fripp last year successfully removed a brooch by œsophagotomy in a child only a year old.

The advantages of treating these cases by the help of the direct-vision œsophageal tubes are:—

(1) The foreign body can be distinctly seen and manipulated either by a hook or by a long pair of forceps.

(2) Under normal circumstances the walls of the œsophagus are in contact, a lumen only existing during the passage of food. When a foreign body is extracted with the help of one of these tubes the walls of the œsophagus are held apart, and instead of being dragged along the œsophageal wall it passes upwards into a definite space. This greatly diminishes the liability to injury.

(3) Since it is possible to see what one is doing a sharp or projecting edge may be drawn under cover of the tube.

(4) The foreign body may be drawn up without any preliminary displacement downwards, as necessarily happens with the coin-catcher or probang. This is of great importance, as the point which is caught in the œsophageal wall naturally will point downwards, and any attempt to push the foreign body downwards will probably produce a laceration.

At the French Surgical Congress in Paris last October Dr. Guisez exhibited about twenty foreign bodies of very varied description which he had extracted from the œsophagus or from a bronchus by means of direct-vision tubes. Of these three were dentures. I had also the opportunity of examining his instruments, which were slightly different from those I have shown to-night. The most important variation was a hinge situated about $1\frac{1}{2}$ in. from the lower end of the tube. By rotating the eye-piece the hinge opened, causing the lower end of the tube to expand and thus making a larger space into which the foreign body could be drawn.

DISCUSSION.

Mr. H. BALDWIN said the method was a great advance on any that had been used before for the purpose of removing foreign bodies from the œsophagus, and he wished to know whether the instrument might not be made with a larger tube, seeing that the œsophagus was the channel down which it had to go and could be dilated to a much larger size. In fact, he thought it possible to have a tube twice the size of that shown. Also, if a tube had an end expanding in all directions, it would afford a much better chance of extracting such bodies as artificial plates with sharp wires. Although the operation had been made by the means described much easier than it used to be, he could not help feeling that a great deal more attention ought to be paid by dentists to the design of plates, especially small plates, in view of the danger should they be accidentally swallowed. He had read a communication before the Odontological Society a short time ago on the subject, and had given a series of designs for small plates showing how almost every kind of small plate could be made, so that if swallowed it would never attach itself to the mucous membrane, there being nothing sharp which could stick. Plates were dangerous chiefly because of the sharp hooks and wires, which were made without the slightest thought about the danger if swallowed. Smooth plates without sharp corners or hooks would probably be swallowed straight into the stomach if swallowed at all.

Mr. STANLEY MUMMERY was especially interested in the method described, and related a case that occurred when he was a dresser at St. George's Hospital. A soldier was admitted who had swallowed a plate with three teeth upon it, and it was located by the X-rays opposite the division of the bronchi. The patient was put under an anæsthetic, and an attempt made to remove it with forceps, but this failed, and œsophagotomy was performed. The plate was removed successfully through the opening, and the case went on very well for two or three days, but after the third day suppuration came on, and while the patient was sitting up in bed one day washing his hands he fell back dead from ulceration through the jugular vein.

Mr. W. RUSHTON said that those who had seen dental newspaper cuttings of recent years must have been struck with the tremendous increase in the

record of cases from swallowing dentures, and also with the large proportion of fatal results ensuing. In connexion with the operation he had never seen suggested what he thought would be of great use in the recovery of plates—namely, some means of dividing a vulcanite plate before extracting it from a patient. He wished to know if any such means had ever come before the notice of Mr. Turner, whether in the shape of cutting forceps which could be manipulated somewhat in the same way as the expanding tube, or whether in the shape of an electric cautery for dividing a vulcanite denture. He agreed with Mr. Baldwin that a grave responsibility rested with dentists in designing plates and also in warning patients as to the risk they ran. As a rule, the cases occurred with a small, ill-fitting or broken plate, either when the patient was asleep or under the influence of intoxicants.

Mr. PHILIP TURNER, in reply, said the tube was a small one, and he had brought it up because it was the one he actually used and the largest he had an opportunity of using at the time. It could be made quite safely half as large again and with expanding ends. He had been interested in hearing of the case quoted by Mr. Stanley Mummery, and he thought that a fatal result happened in a very large proportion of cases of œsophagotomy. With regard to an instrument for dividing the plate, in the case he had to treat the plate was metallic, but he thought a pair of cutting forceps might occasionally be manipulated through the ends of the tube, though it should be remembered that of necessity the tube had to be of great length. He did not think there was any possibility of using an electric cautery, as it would hopelessly damage the walls of the œsophagus.

Radiographs of the Teeth and Associated Parts.

By CHARLES A. CLARK, L.D.S.

I HOPE to show this evening some radiographs of cases demonstrating the uses of X-rays in dental surgery, also mention conditions where their use is at present limited.

Although it was my intention that the *modus operandi* should form no part of this paper, yet I find it is impossible to eliminate it. The method I prefer for taking the teeth is by means of films in the mouth, as there is then no confusion between the two sides of the jaws, as may occur in some positions when a plate is used outside the face even when taken stereoscopically, and I will show you some cases where the film method was the means of diagnosing where the other method had failed. Also fractures of the mandible, odontomes, and tumours generally can be shown with more precision.

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You are probably aware radiographs do not appear at their best as prints or as lantern slides, therefore I have the negatives on view, and am only using the slides to facilitate the explanation of different points of interest. But, before going further, I must thank the Deans of the Royal and National Dental Hospitals for kindly allowing me the use of the negatives of their respective hospitals. An X-ray department was fitted up at the Royal Dental Hospital with the latest apparatus last October, and already as many as 160 patients have been seen there, which shows that X-rays are looked upon as a necessary adjunct in diagnosis in this twentieth century. We have all heard recently a great deal about X-rays dermatitis, and I am very pleased to be able to say that, although I have been radiographing patients for about twelve years, I have not had one case of dermatitis. I think it is important that this should be known, because many patients are deterred from being X-rayed on this account; neither have I any dermatitis myself.

In taking radiographs of the incisive region films are usually best, but it is sometimes as well to take the upper region antero-posteriorly, having the tube a little below the occiput. I find upper centrals are always erupted, while laterals may be entirely absent.

But during the past week I have had two cases of absent centrals in the mandible. The first was that of a young lady aged 25, who had retained her temporary centrals in mandible until a few weeks ago, when one fell out and the other, I presume, being loose, was removed. The radiographs show that there are no centrals. The second case was that of a boy aged 5, son of a medical man. His dentist writes: "There is a strong history in the family of missing permanent teeth. One member, aged 36, has only the two last molars in the lower jaw, the rest being temporary. This little boy's temporary teeth are now decaying rapidly."

I only radiographed the upper and lower incisive region and the lower right premolar region, deferring other radiographs to another visit, as I feared so much exposure might give dermatitis. Besides, it is tiring to so young a patient. The radiographs show erupting 5 4 3 2 | only.

Slide 1.—Region of upper incisors to show length of central right and of two supernumeraries, and if any other teeth or germs present; teeth much distorted.

Slide 2.—Direction of upper centrals, supernumeraries present.

Slide 3.—Dentist was unable to rotate lateral; failure caused by curved root.

Slide 4.—*Re* 1 2 3 region; shows 1 2 3 against small odontome.

Slide 5.—(1) Retarded eruption of lateral; (2) position of root of lateral; different patients.

Slide 6.—Superior laterals, query supernumeraries, as it was hoped, owing to patient's appearance, that there might be normally-sized laterals unerupted, but is simply a case of rudimentary laterals.

Slide 7.—Radiograph of upper incisor region.

Slide 8.—First premolar not erupted, ulcerated cheeks and abscess; patient had much pain; radiograph shows abscess on apex of an apparently sound lateral; lateral was drilled into and pulp found dead. This patient had had this pain for a long time and had been radiographed at a general hospital with plate outside face without finding the cause, subsequently coming on to the National Dental Hospital, and I think this shows the advantage of the films method; all pain disappeared after treatment; cause of dead pulp unknown.

Slide 9.—Patient has a rudimentary lateral on one side and temporary lateral on the other; radiograph shows no permanent lateral below temporary.

Slide 10.—*Re* the roots of the laterals; open apices.

Slide 11.—Central injured by blow, much pain for a long time; radiograph shows absorption of apex.

Slide 12.—Girl aged 16, all teeth regular except upper right lateral, which protruded without any apparent cause, as faulty bite, or pencil biting; supernumerary suspected; radiograph shows no supernumerary, and tooth, although retracted, persisted in protruding on leaving off plate.

Slide 13.—Spreading incisors, query supernumerary; radiograph shows no supernumerary; note suture and bevelled root.

Slide 14.—Two cases of fanning.

Slide 15.—Two other cases of fanning.

Slide 16.—A case of geminated centrals.

Slide 17.—Twisted centrals, query supernumerary. Canines may be said to be always present, both in the maxilla and mandible, but I have one case where the patient had very few permanent teeth, so I will show this before the others.

Slides 18, 19, 20.—Instructions given are: Delayed dentition in upper, 5, 4, 3, 2; both sides are missing; wanted explanation of delay (models). Patient's age, 12; feebly and mentally deficient. Radiographs show all the teeth to be absent in maxilla. In mandible only six-year molars present. Radiographs are not so good as I should like, owing to the difficulty I had with him. Radiographed on two occasions as case was interesting, taking in all fourteen radiographs.

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Slide 21.—Query position of canines; radiograph shows them very high up. On taking the right side (slide 22), which was second to be done, patient's mother persisted in making the child laugh, hence a poor radiograph.

Slide 23.—Crowded buried canines.

Slide 24.—Four cases of buried canines.

Slide 25.—Canine crowded between central and lateral.

Slide 26.—Patient aged 40 or 50; chronic abscess; canine could be just felt with a probe; position supposed to be horizontal. Radiograph shows it to be vertical.

Slide 27.—Patient aged about 20. Horizontal canines; one broken in extraction; position of root required.

We now come to premolars: sometimes absent.

Slide 28.—Second premolars entirely absent; have not been extracted.

Slide 29.—Missing second bicuspid and dilacerated temporary canine.

Slide 30.—Two cases of unerupted premolars for fanning of front teeth; no crowding in one case.

Slide 31.—Similar cases; consider these examples of usefulness of X-rays in study of development.

Slide 32.—Premolar region, three cases.

Slide 33.—Two cases of absent second premolars.

Slide 34.—Query second premolar; is present.

Slide 35.—Ditto two cases.

Slide 36.—Ditto.

Slide 37.—Two cases; buried canine, and missing second bicuspid on one side of mandible.

Slide 38.—Two cases of crowded second bicuspid in mandible.

Slide 39.—Two similar cases to the last.

Slide 40.—Patient had pain in wisdom region, but radiograph shows there is no erupting wisdom.

Slide 41.—Patient had severe neuralgia for ten years. No history of extraction of wisdom tooth. Radiograph shows absence of wisdom and of molar stumps.

Slide 42.—Buried and exostosed stumps.

Slide 43.—Buried temporary stumps between second bicuspid and first molar; cause apparently of abscess in that region.

Slide 44.—Query tooth remnants or necrosis. Radiograph shows no roots present, and alveolus appears normal.

Slide 45.—Patient's age about 50; great pain for two years about

the right side of face; no sleep for more than two hours at a time, and then only by taking drugs. Pain appeared to come from region of first premolar, and certainly I unfortunately produced an attack when holding the film in the mouth against this region. Radiograph shows buried stumps. Whether this was the cause, or whether it was a case of trigeminal neuralgia, I am unable to say, not having, of course, the whole clinical history. This patient had been previously X-rayed by a well-known radiographer with the X-ray plate outside the face without result; again evidence in favour of films.

Slide 46.—Patient lost broken silver spiral tube which could not be felt by probe. Radiograph shows tube, which was subsequently removed easily. Taken on plate outside face.

Slide 47.—Case of difficult extraction of wisdom tooth, subsequently becoming very loose. Query broken posterior root. Radiograph shows large abscess cavity.

Slides 48, 49, 50, 51, 52, 53, 54.—Now we come to cases of Rigg's disease. X-rays are useful here for showing extent of disease, and if radiographs are taken from time to time will show if the disease is arrested or not. Note spot in alveolus and gum margin.

The chief objection to films is not knowing the angle at which the tube is placed, especially in reference to buried canines in the maxilla. This difficulty can be lessened by placing the negative, previously smeared with glycerine or vaseline, in the mouth or on the model; or both methods—films and plate negatives—adopted for the case.

I have attempted, however, to improve upon this by taking the films stereoscopically, and although this can be done, yet the result is a little disappointing for several reasons. First, the negatives are relatively too small compared with the size of the teeth. Also, the anticathode being at the usual distance of 12 in. from the object, moving the tube 3 cm. from the centre is too much and the negatives may be distorted so as to be useless, or, indeed, the point aimed at—especially if this happens to be at the curve of the jaws—may be lost altogether. Perhaps someone here better acquainted with physics than I am could make a suggestion. Probably the distance should be increased to 2 ft. instead of 1 ft. However, in the bicuspid and molar region, the result is an improvement in my opinion.

There is another advantage I may mention. If the radiographs are taken on a whole plate (size) stereoscopically, the dentist will not have a Wheatston stereoscope to put in when he gets them; but with

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stereoscope films they can be placed in an ordinary photographic stereoscope, which he probably has, or if not can get one for one shilling and sixpence or so which will answer his purpose.

One practical difficulty that occurs in X-raying the teeth is in the face powders so generally containing salts of bismuth. Not only is my calculation of penetration but also exposure upset, and to ask a lady to wash her complexion off is impossible; but the difficulty is a real one nevertheless.

I am frequently asked to take radiographs of suspected necrosis. Now I am of opinion that this cannot be definitely determined by means of the rays unless the necrosis is well sequestered. It is entirely a question of radiographic density, and it would be very interesting if the radiographers present would give their experience in this matter.

Then there is another position I have to radiograph which I find very difficult—the temporo-mandibular articulation. I have obtained it sometimes, but cannot make always sure of getting it. Consequently, I asked several of my X-ray friends, but they were unable to help me. But my friend, Dr. Worrall, in radiographing pituitary tumours, frequently also obtained the articulation. I have seen some of these, and have asked him to bring them here this evening, but, owing to an accident, I am sorry he will be prevented.

Thickened mucous membrane in any of the sinuses cannot now be definitely determined. In cases of the maxillary antrum a difference may be seen, but, as was stated at a recent meeting of the Electro-Therapeutical Section when this matter was discussed, it cannot be definitely diagnosed.

DISCUSSION.

The PRESIDENT (Mr. J. Howard Mummery) wished to know what special precautions Mr. Clark took to prevent dermatitis. From a recent discussion in the Electro-Therapeutical Section it appeared that some people were exceedingly susceptible to the X-rays. This especially applied to the operator, who was frequently exposed to them, and therefore it would seem special precautions were desirable. He also desired to know whether Mr. Clark could explain the triangular space seen at the symphysis of the mandible, and which was shown in many of the negatives upon the table. He asked this question as this appearance had been looked upon as pathological in a case in practice, but he found it was very frequently seen in X-ray negatives of the normal jaw.

Mr. DEANE BUTCHER tendered his thanks from a radiologist's point of view to Mr. Clark for his most admirable and interesting series of dental skiagrams.

On the question of technique he inquired whether a soft or hard tube was used, and what was the length of the exposure? He was pleased to hear of the success of the photographic film, as he had always thought there might be some advantage in the use of a film in contact with the skin for the Röntgen examination of the kidneys and other internal organs. With regard to the supposed danger to patients or operators, it was true that very regrettable accidents occurred in the early days of Röntgen photography. That danger was past, and it had been truly said that at the present day "the man who burnt his patient was a knave, and the man who burnt himself was a fool." As regards the patient, he was as safe in the hands of a radiologist as he was in a dentist's chair, and far more comfortable. He thought few people recognized what a large amount of labour, skill, and training was required for the production of such a series of pictures as Mr. Clark had shown.

Dr. HOWARD PIRIE expressed admiration at the photographs Mr. Clark had shown, and thought the stereoscopic effect would be improved if a wire were wound round the teeth that were going to be photographed, the wire projecting slightly inwards and slightly outwards. That gave a sense of depth and guided the eyes to see one part well in front of the teeth and one part well behind. The same effect was obtained in looking at the telegraph wires on the side of a railway; if the eyes were turned parallel to the telegraph wires it was very difficult to distinguish which wires were in front and which behind, but directly the head was turned at right angles to the wires they stood out quite distinctly. Films produced the best effect with regard to X-ray photographs, but at the same time some good effects could be obtained with a plate on the outside of the face. To show this Dr. Pirie exhibited slides taken from plates on the outside of the face. One showed stereoscopically a fractured jaw, and the other a supernumerary tooth blocking the way of an unerupted incisor tooth. The latter condition had been diagnosed by the stereoscopic method, and the supernumerary was extracted by Mr. J. G. Turner. He also showed a slide of central incisor teeth. At the same time he thought that films were much the easiest, and, as a rule, the best.

Mr. SIDNEY SPOKES had recognized three amongst Mr. Clark's photographs in which he was himself interested. None of them had the bismuth trouble that had been referred to. Most of the cases he had asked Mr. Clark to radiograph had been in connexion with buried canines. The most interesting one was that which showed a very much broken-down root of a bicuspid tooth underneath a bridge in the mouth of a gentleman who had suffered pain for two years. He had had the bridge for some time and had been told there was nothing underneath that need give him any trouble. An endeavour was made to find the presence of the root before taking the bridge off, but he was not able to satisfy himself exactly what there was there. Mr. Clark's radiograph, however, showed exactly where the bicuspid root was. The bridge was taken off and the buried root removed, the radiogram being of extreme use in the treatment of the case, as it was necessary to remove alveolus in order to reach the fragment.

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Dr. FINZI said the method he employed was to use films inside the mouth, but at a distance from the tube of a metre in order to prevent distortion, and by using an intense current he could reduce the exposure to less than two seconds, so that the patient was able to keep quite still during the time. The definition obtained was really remarkable, as the slides which he had with him would show. Dr. Finzi exhibited four slides illustrating his remarks.

Mr. W. HERN asked Mr. Clark whether he could work out some method whereby stereoscopic slides could be obtained, because it was just with regard to irregularities of the teeth that the stereoscopic principle would be so valuable. Some time ago Mr. Mackenzie Davidson had shown him some excellent photographs of shots in the eye where by the use of stereoscopic vision the shots could be located at once. One photograph showed a bullet lying either in front of or behind the interosseous membrane, and when asked where the bullet was he, without the stereoscope, thought it was in front, but when the photograph was put into the stereoscope it could at once be seen to be immediately behind. If that stereoscopic method could be worked out for dental surgery it would be exceedingly valuable for locating the exact positions of misplaced teeth or buried roots. He agreed with Mr. Clark that small films placed in the mouth gave greater definition than the ordinary photographs taken through the face.

Mr. CLARK, in reply, said with regard to X-ray dermatitis, the great thing was not to expose the patient too long to the rays, and he avoided screening as much as possible. Also he believed that fair patients were more liable to dermatitis than dark. With regard to the light space in the chin he was sorry to say he had not noticed that before, but as in all the negatives exhibited where the chin was shown the space was to be seen, he thought therefore it must be normal. With reference to exposure, in ordinary work he generally gave just under ten seconds, but at the Royal Dental Hospital he could reduce it to anything he liked. A very rapid exposure was a strain upon the tube. The tube used was an ordinary one, and for teeth the spark gap should be about three inches. As to the use of wire on the teeth, for stereoscopic purposes it was a little troublesome, but in the cases he had tried he obtained better results. He agreed that a better definition was obtained if the tube was at a distance of about two yards, and with very short exposures. With regard to Mr. Hern's question as to the stereoscopic method, possibly he had not seen that there were instruments in the room in which the stereoscopic effect was very well shown.

The retiring PRESIDENT (Mr. J. Howard Mummery) delivered a valedictory address, and a vote of thanks for his services was carried by acclamation.

Odontological Section.

February 22, 1909.

Mr. LEONARD MATHESON, President of the Section, in the Chair.

Specimens added to the Museum.

By J. F. COLYER, M.R.C.S., L.D.S.

THE HON. CURATOR said the period he had to report upon covered eighteen months, and during that time 180 specimens had been added to the museum, and of those there were roughly about forty specimens of comparative dental pathology. Amongst the anatomical specimens that had been added to the museum there were about fifty skulls of dogs the breeds of which were known. He proposed during the coming year to rearrange and classify the section dealing with irregularities of the teeth. There were very few anatomical specimens illustrating that section, and he should be pleased if any members who happened to have skulls illustrating irregularities in the position of teeth would present them to the museum.

The Curator stated that since the last meeting the following specimens had been added to the collection :—

(1) The skull of a pig-tailed monkey showing the absence of the mandibular incisor teeth. The specimen had been radiographed and the radiograph showed an absence of the incisors (figs. 1 and 2).

(2) The skull of a Scotch collie with marked destruction of the enamel of many of the teeth, together with a very definite carious cavity in the mandibular left second molar (fig. 3). There was no history of the case, but there was a similar specimen in the museum with a very definite history of prolonged distemper, and there was very little doubt that the destruction of the tooth tissue was due to the lodgment of food and should be regarded as true caries. There was a thickening of the margin of the alveolar process, probably due to a marginal gingivitis.

(3) The skull of a pug dog (fig. 4), the interesting feature of which was the marked wear of the incisor teeth. The margins of all the incisor and canine teeth and the cusps of the carnassial teeth showed marked attrition. The maxillary right first molar had been the seat of suppurative periodontitis, and the second molar had been lost. The periodontal



FIG. 1.

Mandible of pig-tailed monkey,
showing absence of incisors.



FIG. 2.

Radiograph of mandible of pig-tailed
monkey, showing absence of incisors.



FIG. 3.

Mandible of Scotch collie, showing destruction of enamel and carious cavity
in the left second molar.

disease had gone on to the destruction of practically the whole of the bone in the neighbourhood of the tooth. It was interesting to note that the attrition on the carnassial teeth was more marked on the healthy

side. The skull was also of interest because it showed four extra premolars, one on either side of the maxilla and one on either side of the mandible.

(4) The skull of a Vervet monkey (fig. 5) showing marked thickening of the bones of the face and skull, the result of "rickets." The monkey

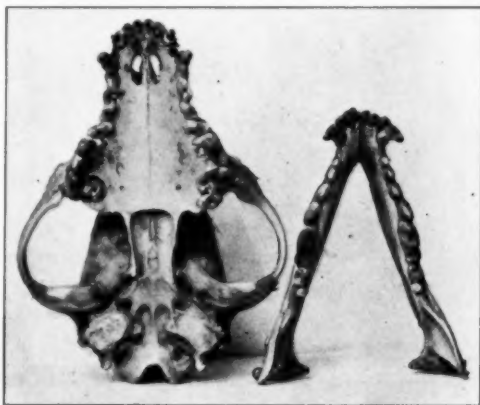


FIG. 4.

Skull and mandible of pug-dog, showing marked attrition of teeth and destruction of bone in region of right maxillary molars from periodontal disease.



a

FIG. 5.

b

a, Rickety skull ; *b*, normal skull.

was nearly an adult animal, and it was interesting to note that as far as the arch of the teeth went there was not the slightest displacement of the teeth, the occlusion of the premolars and molars being good. As far

as he could make out, when monkeys had "rickets" the disease did not seem to interfere with the dental arch, whereas with lions and tigers with rickets there was often a very marked displacement of the teeth.

(5) The skull of a dasyure (fig. 6) showing marked destruction of the alveolar process in the mandibular molar region, the result of periodontal disease.



FIG. 6.

Mandible of a dasyure, showing alveolar process from periodontal disease.

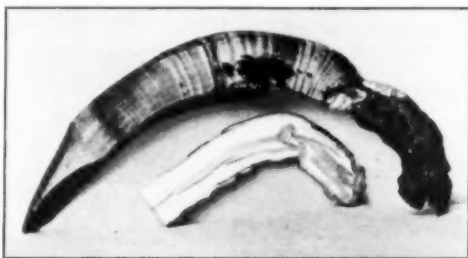


FIG. 7.

Tusk of wild boar, showing results of injury.

(6) A portion of the mandible of a bear in which the left canine, left third incisor, and first premolar had been lost as a result of severe injury. These teeth had been either shot or torn away, and there had been a good deal of suppuration. The bone was considerably thickened and showed signs of having been the seat of extensive suppuration. It was a typical example of the wonderful amount of injury an animal was able to endure and yet live.

(7) A skull with a cyst invading the antrum.

(8) The tusk of a wild boar that had been injured (fig. 7). About 2 in. from the end of the tooth the regular formation of the enamel ceased, the appearance of the tooth beyond suggesting that the enamel organ had been injured and had formed enamel in a sporadic



b *a*
FIG. 8.

a, Specimen showing fusion of second and third maxillary molars;
b, Section through *a*.



FIG. 9.

Right maxilla of an individual about twenty years of age, showing misplaced third molar and permanent canine.

manner. About an inch from the end of the tooth there was a marked kink filled with cementum, that tissue also forming a thick covering over the distal 2 in. of the tooth. It showed how animals whose teeth grew from persistent pulps could overcome a great deal of injury to those pulps.

(9) A specimen shows gemination between the second and third molars (fig. 8).

(10) The maxilla of an individual, about twenty years of age, showing misplaced third molars and canines (figs. 9 and 10).



FIG. 10.

Radiograph of specimen shown in Fig. 9, showing relation of misplaced third molar to the antrum.

The PRESIDENT (Mr. Leonard Matheson), in moving the adoption of the report of the Hon. Curator, paid a tribute to Mr. Colyer's work in connexion with the museum and said the Section was not only proud of its museum, but proud too of its Curator.

General Results of the Special Rules in force in Match Factories.

By KENNETH W. GOADBY, L.D.S., D.P.H.

IN March, 1907, I was requested by the Secretary of State for the Home Department to make inquiry into the working of the Special Rules for lucifer match factories in which yellow phosphorus is used, the Home Secretary stating that "The Special Rules are believed to have lessened materially the risk of phosphorus necrosis, but a fatal case occurred in 1905, and another has been reported this year, both from the same factory."

It will be remembered that in 1899 Mr. George Cunningham, Professor Sir Thomas Oliver, and Professor Thorpe, issued a joint Report¹ on the use of phosphorus in the manufacture of lucifer matches, and that subsequent to this report a public inquiry was held in which the question of the treatment of the workers in phosphorus was exhaustively inquired into. The Special Rules which were afterwards laid down for the conduct of lucifer match factories where white phosphorus is used were based on the knowledge obtained during the inquiry that yellow phosphorus was the compound associated with poisoning, whereas red or amorphous phosphorus, such as is used in the production of safety matches, did not appear to be associated with any poisoning. The Special Rules, therefore, apply only to the manufacture of matches containing yellow phosphorus, such matches being the well-known "strike anywhere" match.

The Special Rules contain provisions relating to the ventilation of the workshops, dipping, stoves, &c., regular medical and particularly dental inspection, the examining Dental Surgeon being appointed by the individual firm and the appointment submitted to the Home Office for confirmation.

The Special Rules Nos. 5, 6, 7, and 8, are those which especially relate to the Dental Surgeon, and are the ones upon which I was requested to obtain precise information.

¹ Reports on the use of Phosphorus in the Manufacture of Lucifer Matches by Professor T. E. Thorpe, L.L.D., F.R.S., Professor Thomas Oliver, M.D., F.R.C.P., Mr. George Cunningham, 1899. [C9188.]

SPECIAL RULES FOR LUCIFER MATCH FACTORIES WHERE WHITE
PHOSPHORUS IS USED.

No. 5.—(a) For the purposes of these Rules the occupier shall appoint, subject to the approval of the Chief Inspector, a duly qualified and registered dentist, herein termed the appointed dentist. It shall be the duty of the appointed dentist to suspend from employment in any phosphorus process any person whom he finds to incur danger of phosphorus necrosis by reason of defective conditions of teeth or exposure of the jaw. (b) No person shall be newly employed in a dipping room for more than twenty-eight days, whether such days are consecutive or not, without being examined by the appointed dentist. (c) Every person employed in a phosphorus process, except persons employed only as boxers of wax vestas or other thoroughly dry matches, shall be examined by the appointed dentist at least once in every three months. (d) Any person employed in the factory complaining of toothache, or a pain or swelling of the jaw, shall at once be examined by the appointed dentist. (e) When the appointed dentist has reason to believe that any person employed in the factory is suffering from inflammation or necrosis of the jaw, or is in such a state of health as to incur danger of phosphorus necrosis, he shall at once direct the attention of the certifying surgeon and occupier to the case. Thereupon such person shall at once be examined by the certifying surgeon.

No. 6.—No person shall be employed in a phosphorus process after suspension by the appointed dentist; or after the extraction of a tooth; or after any operation involving exposure of the jaw-bone; or after inflammation or necrosis of the jaw; or after examination by the appointed dentist in pursuance of the Rule 5 (d); or after reference to the certifying surgeon in pursuance of Rule 5 (e), unless a certificate of fitness has been given, after examination, by signed entry in the Health Register, by the appointed dentist or by the certifying surgeon in cases referred to him under Rule 5 (e).

No. 7.—A Health Register, in a form approved by the Chief Inspector of Factories, shall be kept by the occupier, and shall contain a complete list of all persons employed in each phosphorus process, specifying with regard to each such person the full name, address, age when first employed, and date of first employment. The certifying surgeon will enter in the Health Register the dates and results of his examinations of persons employed in phosphorus processes, and particulars of any

directions given by him. The appointed dentist will enter in the Health Register the date and results of his examination of the teeth of persons employed in phosphorus processes, and particulars of any directions given by him, and a note of any case referred by him to the certifying surgeon. The Health Register shall be produced at any time when required by H.M. Inspectors of Factories, or by the certifying surgeon, or by the appointed dentist.

No. 8.—Except persons whose names are on the Health Register mentioned in Rule 7, and in respect of whom certificates of fitness shall have been granted, no person shall be newly employed in any phosphorus process for more than twenty-eight days, whether such days are consecutive or not, without a certificate of fitness granted after examination by the certifying surgeon, by signed entry in the Health Register. This rule shall not apply to persons employed only as boxers of wax vestas or other thoroughly dry matches.

The statistics and general evidence set out in the present paper deal exclusively with the above rules in so far as they apply to the dental condition of the workers.

Ventilation, however, is especially a most important question, and there is little doubt that the increased vigilance directed to ventilation in all dangerous processes is a potent factor in the diminution of the risk of phosphorus poisoning, and it may be thought by some that the certain decrease in phosphorus poisoning that has taken place since the introduction of the rules is more related to the improvement in ventilation than to the Special Dental Rules. In the course of my inquiries, however, I did not find any especial relation existing between the cases of poisoning reported and the general efficiency of the ventilation plant. It is true that the standard varied in different factories, but the variation had not the same relationship to the poisoning that the dental condition had; moreover, in factory "I" in the tables, the ventilation was better than in certain other factories, and a good deal of attention had been paid to the question of exhaust ventilation for some time at this factory. It must not be thought that I am minimizing the importance of ventilation; I only point out that, given efficient ventilation and a bad dental condition of the workers' mouths, poisoning may take place.

My terms of reference suggested the determination of precise information under the following headings:—

(1) The adequacy of the present rules so far as the system of periodical dental examination is concerned, and the precise improvements, if any, which can be suggested.

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(2) The amount and character of the dental work accomplished.

(3) The principles which the appointed dentists have laid down for their own guidance, as, for example, the duration of suspension after extraction, and how far the entries in the Health Register show that they have been carried out.

(4) Instances of failure of Rules 5, 6, and 7 to meet danger to workmen of contracting phosphorus necrosis. I shall not attempt to discuss all these points, but to give a general résumé of my report.¹

At the time of my visits to the lucifer match factories in 1907, nine factories employing 2,586 hands were in operation, and of these 902

TABLE I.—PERSONS EMPLOYED AND EXAMINED.

Factory	Names on Health Registers, 1900-1907	PERSONS EMPLOYED AT TIME OF SPECIAL EXAMINATION				PERSONS INCLUDED IN SPECIAL EXAMINATION.			
		In phosphorus processes		In other processes	Total employed	In phosphorus processes		In other processes	Total examined
		Examined under Rule 5c	Other			Examined under Rule 5c	Other		
(1)	(2)	(3)	(4)	(5)		(7)	(8)	(9)	(10)
A	847	22	171	440	633	22	34	—	56
B	89	10	21	58	89	10	21	—	31
C	903	7	29	34	70	7	29	—	36
D	1,572	512	—	410	922	159	—	9	168
E	150	33	4	—	37	33	2	—	35
F	82	3	20	—	23	3	20	—	23
G	72	1	8	17	26	1	8	—	9
H	332	7	90	59	156	7	90	37	134
I	1,378	307	—	323	630	307 ¹	—	65	372
	5,395	902	343	1,341	2,586	549	204	111	864

¹ Including 258 dry match boxers and frame fillers who have been examined monthly since January, 1907, but subjected to no treatment except occasional extraction.

were regularly examined by the appointed dentist under Rule 5 (c). But of this number 512 were present in one factory alone. The totals of the numbers employed, and those examined by the appointed dentist under Rule 5 (c), and those at work in other processes, will be found tabulated in Table I. It was difficult to obtain the total number of persons who had passed through the various factories, as in many cases the persons employed in boxing dry wax vestas or other dry matches, and who,

¹ Special Report on the Working of Rules in Lucifer Match Factories. Report of H.M. Chief Inspector of Factories and Workshops, 1907.

according to the Special Rules, are not examined by the appointed dentist unless they complain of toothache or other dental trouble (Rule 5 (*d*)), were, in a large number of cases, not recorded in the Health Registers. The total therefore of 5,395 which appeared in the Health Registers between 1900 and 1907 does not represent the entire number of workers in all processes during these periods, but includes all who worked in dangerous processes. Of the number of persons (2,586) employed in the various factories, I examined the mouths of 864 persons. In the case of Factory D, 159 only of the total number 512 were examined. Here the dental conditions were particularly good. But in Factory I, where the recent fatal case had occurred, and where the dental conditions were very bad, almost the whole of the factory were examined, including the very large number of persons (258) employed in boxing dry matches or frame filling.

The method of examination was as follows: Each worker's mouth was examined for the presence of (*a*) carious cavities, (*b*) fillings, (*c*) recent or old extractions, (*d*) erupting teeth, (*e*) evidence of inflammatory conditions.

The results were recorded on charts in a large loose-leaf diary—samples of these charts I will pass round—the charts were rapidly marked with blue pencil, and at the conclusion of the examination tabulation was made on each chart of the various conditions of the mouths found. At each factory the Health Registers were examined, and the total number of persons entered on the registers as having been treated under Rule 5 (*c*) were tabulated, together with the number of fillings, extractions, and so on. These figures are set out in Table II which gives the work performed by the various appointed dentists between the institution of the Rules in 1900 and the time of the examination. The figures are given for the separate factories.

In Table III the condition of the mouths of persons employed in phosphorus processes, and examined regularly under Rule 5 (*c*) by the appointed dentist, are set out.

The number of persons examined in the course of my inquiry was 864; of these 549 were examined quarterly by the appointed dentists, but of this number 258 employed at Factory I have only been included in the general examination since January, 1907, and then only for the purpose of determining if any of the members examined had returned to their work after recent tooth extraction. Only 49 persons from Factory I are properly included in persons examined by the appointed dentist under Rule 5 (*c*). The total number therefore of persons regularly examined at the quarterly examination by the appointed dentist

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TABLE II.—WORK PERFORMED BY APPOINTED DENTISTS.

Factory (1)	Persons examined under Rule 5c, 1900 to 1907 (2)	Fillings (3)	EXTRACTIONS		Scaling and gum-cleansing operations (6)	Suspensions by certifying surgeons (7)	Reported cases of phosphorus poisoning, 1900 to 1907 (8)
			Total (4)	With anesthetics (5)			
A	115 ¹	93	80	60	20	20	—
B	14	—	—	—	—	—	—
C	42	38	36	9	—	4	—
D	1,572 ²	6,281	2,728	988	1,347	—	—
E	150	206	142	34	1	1 ³	2
F ⁴	9	1	6	?	?	—	—
G ⁵	10	89	41	8	—	—	—
H	33	25	97	33	5	—	—
I	160	320	58	—	—	4	6 ⁶
	2,105	7,053	3,188	1,082	1,373	29	8

¹ Boxers not examined.² Boxers examined and treated when necessary.³ Certified as necrosis.⁴ Commenced in 1902.⁵ Partially closed at time of examination pending erection of new machines.⁶ Two of these cases were fatal. Since this report was written two other cases have been reported from this factory.TABLE III.—CONDITION OF MOUTHS OF PERSONS EMPLOYED IN PHOSPHORUS PROCESSES¹
EXAMINED UNDER RULE 5c.²

Factory (1)	Healthy mouths (2)	Teeth missing (3)	Fillings present (4)	Teeth requiring extraction (5)	Inflammation of the gums (6)	Suppuration about the jaws (7)	Teeth requiring filling (8)
A	16	45	11	—	—	—	9
B	3	43	1	24	3	—	5
C	3	48	13	6	1	—	1
D	109	751	572	—	3	—	58
E	4	202	30	92	10	1	3
F	1	18	2	4	1	—	—
G	—	14	11	—	1	—	—
H	5	51	16	—	5	1	3
I ³	1	103	105	90	16	5	26
	142	1,270	761	216	40	7	105

¹ Mixers, dippers, dippers' helps, carriers away, drying-room hands, cutters down, and wet match boxers.² See Table I, column 7.³ The details for this factory do not relate to the 258 boxers and frame fillers (see footnote to Table I and Table IV).

under Rule 5 (c) is 291, and of this number 142, or 48·8 per cent., had healthy mouths in so far as no carious teeth were present, or inflammation of the gums, though several had teeth missing, and there was therefore no evidence of dental trouble predisposing to phosphorus necrosis. Of this number of healthy mouths (142), 109 were accounted for by one factory alone, whereas in another factory only two persons had sound mouths. And in this same factory, of 49 persons engaged in wet process four had distinct suppuration with sinuses leading down to bare bone. All these four persons were regularly examined under the Special Rules as they were working in a dipping house, where a recent fatal case had occurred, and had been passed as sound by the appointed dentist and entered by him as free from dental disease in the Health Register.

In Table IV I have given the numbers of the persons examined for various age groups, dividing the cases into the male and female for each factory, and into those examined by the appointed dentists and those not examined by the appointed dentists, whereas the percentages for each factory are given in Table V.

In Tables VI and VII the percentages of persons are collected from the various factories and divided into the four classes suggested by Mr. Cunningham in his report. The criteria adopted by him were as follows:—

Good.—Where all permanent teeth were sound, or were artificially made sound.

Fair.—Where one, three, or four permanent teeth are decayed or lost.

Bad.—Where five, six, seven, or eight permanent teeth are decayed or lost.

Very Bad.—Where nine or more permanent teeth are decayed or lost.

By this test, however, a person whose mouth had lost nine or ten teeth but in which the gums were entirely healed would figure as very bad, whilst a person who had only two decayed teeth, although there might be distinct suppuration around such roots, might still be classed as fair. In order to make my figures comparable to those of Mr. Cunningham I have used his four classes, but I have used also an extra test (inflammation of the gums), and included as very bad any case showing inflammatory condition of the gums or alveolar process. My table is therefore more stringent than that given by Mr. Cunningham, but at the same time it will be noticed that, notwithstanding the extra stringency of the tests, the figures given show a very distinct improvement in the numbers of good mouths from those given in Mr. Cunningham's list, even among those persons not regularly examined by the appointed dentists.

TABLE IV.—CONDITION OF MOUTHS OF PERSONS EMPLOYED : AGE AND SEX.

3. CONDITION OF MOUTHS OF PERSONS.																						
Factory	Persons examined (2)	Under 18 years				18 to 21 years				21 to 30 years				30 to 40 years				Over 40 years				
		G. (3)	F. (4)	B. (5)	V.B. (6)	G. (7)	F. (8)	B. (9)	V.B. (10)	G. (11)	F. (12)	B. (13)	V.B. (14)	G. (15)	F. (16)	B. (17)	V.B. (18)	G. (19)	F. (20)	B. (21)	V.B. (22)	
(1)																						
A	1 Rule 5c { M. 22 F. —	—	—	—	—	5	1	—	—	4	1	—	—	5	3	—	—	2	—	—	1	—
	2 Other { M. — F. 34	—	13	—	1	1	12	2	—	—	—	—	—	—	—	—	—	—	—	—	—	—
B	1 Rule 5c { M. 6 F. 4	2	—	—	—	—	—	—	—	—	—	1	—	—	—	—	—	1	—	1	—	2
	2 Other { M. — F. 21	—	—	1	—	—	—	—	—	2	5	5	3	—	—	—	—	—	—	—	—	—
C	1 Rule 5c { M. 7 F. —	—	—	—	—	—	—	1	—	2	—	—	1	1	1	—	—	—	—	—	—	—
	2 Other { M. — F. 29	—	3	2	—	—	6	8	2	1	—	2	3	—	—	—	—	—	—	—	—	1
D	1 Rule 5c { M. 48 F. 111	6	7	—	—	2	19	—	—	17	3	—	—	8	3	—	—	2	—	—	—	—
	2 Other { M. — F. 9	—	9	—	—	27	—	—	—	25	9	—	—	—	—	—	—	—	—	—	—	—
E	1 Rule 5c { M. 10 F. 23	—	—	1	—	3	1	1	—	1	1	1	4	—	—	—	—	1	—	1	—	—
	2 Other { M. — F. —	—	—	—	2	—	—	2	5	1	1	2	—	—	—	—	—	—	—	—	—	—

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TABLE V.—CONDITION OF MOUTHS OF PERSONS EMPLOYED: PERCENTAGES AT EACH FACTORY.

Factory		Persons examined	PERCENTAGE			
			Good	Fair	Bad	Very bad
(1)		(2)	(3)	(4)	(5)	(6)
A	...	¹ Rule 5c { M. ...	72.7	22.7	4.5	—
		{ F. ...	—	—	—	—
		² Other { M. ...	17.6	73.5	5.8	2.9
B	...	{ F. ...	—	—	—	—
		{ M. ...	—	—	—	100.0
		² Other { F. ...	9.5	23.3	28.5	38.0
C	...	{ M. ...	42.8	14.2	—	42.8
		{ F. ...	—	—	—	—
		² Other { M. ...	3.4	31.0	41.3	24.1
D	...	{ F. ...	—	—	—	—
		{ M. ...	—	—	—	—
		² Other { F. ...	—	—	—	—
E	...	{ M. ...	50.0	30.0	20.0	—
		{ F. ...	4.3	4.3	21.7	69.5
		² Other { M. ...	—	—	—	100.0
F	...	{ F. ...	—	—	—	—
		{ M. ...	—	—	—	—
		² Other { F. ...	—	5.0	45.0	50.0
G	...	{ M. ...	—	100.0	—	—
		{ F. ...	—	—	—	—
		² Other { M. ...	—	—	—	—
H	...	{ F. ...	—	—	—	—
		{ M. ...	—	—	—	—
		² Other { F. ...	—	37.5	12.5	50.0
I	...	{ M. ...	71.4	—	—	28.5
		{ F. ...	—	—	—	—
		² Other { M. ...	20.0	—	60.0	20.0
J	...	{ F. ...	0.8	19.6	31.9	47.5
		{ M. ...	10.2	22.2	12.2	55.1
		² Other { F. ...	1.5	23.2	27.8	47.2
K	...	{ M. ...	8.3	—	58.3	33.3
		{ F. ...	1.8	24.5	37.7	35.8

¹ i.e., persons examined quarterly by appointed dentists under Rule 5c.

² Including those employed in non-phosphorus processes.

TABLE VI.—CONDITION OF MOUTHS OF 864 PERSONS EMPLOYED IN NINE FACTORIES; AGE AND PERCENTAGES.

Persons examined				PERCENTAGE			
				Good	Fair	Bad	Very bad
(1)				(2)	(3)	(4)	(5)
Aged under 18	Rule 5c	M. 27		37.0	40.7	7.4	14.8
		F. 125		20.8	32.0	26.4	20.8
	Other	M. 6		—	—	50.0	50.0
		F. 118		6.8	38.9	32.2	22.0
Aged over 18	Rule 5c	M. 126		46.0	23.0	8.7	22.2
		F. 271		15.8	21.4	16.2	43.1
	Other	M. 13		15.4	—	53.8	30.8
		F. 178		2.2	25.8	24.7	65.1
All ages	Rule 5c	M. 153		44.4	26.2	8.4	20.9
		F. 396		19.9	24.7	19.4	36.8
	Other	M. 19		10.5	—	52.0	36.8
		F. 296		4.0	31.0	29.3	35.4

TABLE VII.—CONDITION OF MOUTHS OF 965 PERSONS EMPLOYED IN FIVE ENGLISH FACTORIES¹ (FROM MR. CUNNINGHAM'S REPORT).

Process and persons examined				PERCENTAGE			
				Good	Fair	Bad	Very bad
(1)				(2)	(3)	(4)	(5)
Aged under 18	Phosphorus	M. 199		11.6	39.7	32.6	16.1
		F. 318		2.8	24.2	35.9	37.1
	No phosphorus used	M. 46		4.3	21.7	37.0	37.0
		F. 48		2.1	22.9	33.3	41.7
Aged over 18	Phosphorus	M. 93		6.5	24.7	32.3	36.5
		F. 173		1.2	7.5	23.7	67.6
	No phosphorus used	M. 59		1.7	16.9	11.9	69.5
		F. 29		—	3.5	6.9	89.6
All ages	Phosphorus	M. 292		9.9	34.9	32.6	22.6
		F. 491		2.2	18.3	31.6	47.9
	No phosphorus used	M. 105		2.9	19.0	22.9	55.2
		F. 77		1.3	15.6	23.4	59.7

¹ Boxers of dry matches are included in phosphorus processes, but in Table VI they are included under "other."

The percentage of persons with sound mouths that I found working in phosphorus processes and examined regularly under Rule 5 (c) is 44·4 per cent. for males and 17·7 per cent. for females, whereas the percentage of sound mouths noticed by Mr. Cunningham were 9·9 per cent. for males and 2·2 per cent. for females—that is, an improvement since the institution of the special rules and dental treatment of 34·5 per cent. for males and 15·5 per cent. for females, coincidently with a decrease in the cases of poisoning. But, besides persons under supervision it will be seen that a distinct improvement has taken place amongst those persons who were not subject to regular examination. This is either owing to the moral effect of a proportion undergoing dental supervision causing the remainder to take greater care of their teeth, or that a process of selection has gone on ; but whatever the cause a distinct improvement had taken place.

This marked improvement in the dental condition of persons employed in phosphorus processes is more marked in certain factories than in others, and as will be seen on reference to Table V the percentage of good mouths in the various factories varies from 72 per cent. for males and 66 per cent. for females, to 10·2 per cent. for males and 1·5 per cent. for females. This improvement will be seen to be co-related with the amount of attention which was given by the Dental Surgeon, as will be seen on reference to Table II, where the amount of the work performed is set out. It must not be taken, however, that in Factory A the number of suspensions given in column 7 represents suspensions for threatened necrosis, for in Factory A alone the number of suspensions are for unhealed extractions, and after fillings had been inserted, the Certifying Surgeon in this factory desiring to see all cases (extractions and fillings) before they returned to work. It will be seen that in Factory D, although a very large number of extractions were performed, no note at all could be found in the Health Register of the person suspended, but it was invariably the custom in this factory to suspend all workers after extractions until the mouth had entirely healed, the worker having to pass not only the appointed dentist, but the factory doctor as well before resuming work.

From the amount of work performed as noted in the Health Register it would be suggested that the persons in Factory I were in good dental condition. But unfortunately this was not brought out as the result of my personal examination, as will be seen on reference to Table III, where it will be seen that amongst the number of persons examined only one out of forty-nine had a sound mouth, sixteen showed

inflammation of the gums, five showed suppuration about the jaws, and a number of the fillings noted in the Health Register and included in Table II were not only found to have already failed, but in many cases to have entirely disappeared, and the teeth into which they had been inserted to save were represented by carious and decomposing roots. This Factory I is the one in which five out of eleven cases occurred which have been reported since the institution of the rules in 1900. Contrasting this factory with any one of the other factories except where two cases of phosphorus necrosis had been reported, and in whose workmen's mouths at the time of my examination a large number of teeth required filling or extraction, and in which inflammation of the gums was present, it is seen that where the supervision has been efficient no phosphorus necrosis has been reported. In fact, it is shown from the tables that the factory in which the dental condition of the workers was the worst, and presumably had been the worst during the whole period of the working of the Special Rules, was the one in which the largest amount of phosphorus necrosis had taken place. (See Tables II and III.)

The fact is significant, for amongst 1,378 persons whose names appeared on the Health Register between 1900 and 1907 in this factory, six cases, two of them fatal, had taken place, whereas amongst the workers, 4,017 in number, in the other eight factories only two cases had occurred, the remaining four cases being distributed amongst a factory which is not now under the Special Rules, and was therefore not visited, as no yellow phosphorus is used—and a factory that had been closed for some time. Previous to the institution of the Special Rules between 1893 and 1899, thirty-seven cases of phosphorus poisoning had occurred in 1,908 persons employed in dangerous processes as against thirteen cases from 1900 to 1907 in 1,378 persons. There is, I think, no other such striking instance of the good result of the application of special knowledge to industrial disease.

What is brought out very strongly, and is, I think, a matter of considerable congratulation to those who were responsible for the institution of the Special Rules, is that a marked decrease has taken place in the amount of phosphorus poisoning concomitantly with an improvement in the dental condition of the workers in the various factories.

Other countries at the present time who employ phosphorus in the yellow form for matchmaking have a very large amount of poisoning, notably Austria-Hungary. Quite recently in a book published, Dr. Teleky states that in Austria-Hungary in 1902 there were seventy factories for

the manufacture of lucifer matches which employed 4,611 persons as compared with the 4,150 in Great Britain. The factory inspector reported seventy-five cases of necrosis having occurred during 1896 and 1906, but Dr. L. Teleky¹ and his wife, by careful inquiry, obtained definite proofs of 160 cases of necrosis which occurred in only twenty-five of these seventy factories, so that a very large number of cases still occur.

Dr. Teleky and Dr. Gilbert (the principal medical inspector of factories in Belgium) both insist very particularly upon the danger of chronic periostitis and other forms of dental inflammation in addition to the danger caused by dental caries. The dental supervision in these factories is practically *nil*.

In the course of my own inquiries I found that it was by no means an uncommon thing to find persons suffering from swollen face, yet working in a match factory not in the scheduled dangerous processes, but in the boxing of dry lucifer matches.

In conclusion, I should like to refer briefly to the pathology of phosphorus poisoning. Besides the affection of the jaw—phosphorus necrosis or “phossy jaw”—a considerable number of cases of fragilitas ossium occur among the cases of necrosis of the jaw; in fact, in the Belgian match factories Sir T. Oliver states that thirty cases of spontaneous fracture of the long bones occurred in five years. Apparently there is some factor at work which operates on the bones of the body as a whole, and the effect on the jaw would then be only a special case of the general intoxication. I could obtain no evidence among the workers in phosphorus factories of a greater incidence of tuberculosis, nor do the Certifying Surgeons’ reports suggest it. There is also considerable disagreement among various observers as to the existence of a phosphorus poisoning with constitutional symptoms other than necrosis. Armand says 50 per cent. of persons exposed to phosphorous fumes become anæmic and are more liable to bronchitis and dyspepsia than workers in other industries. These figures are almost impossible to obtain, the majority of persons working in match factories being young women who are notoriously liable to dyspepsia and anæmia in other than phosphorus factory work.

During my inspection I noticed that where phosphorous fumes were most noticeable and were evidently commonly present from the construction and arrangement of the workshops the workers so exposed showed such signs of anæmia as loss of brightness of the eye, pale conjunctivæ; but I was unable to make any direct blood examinations.

¹ “Die Phosphornekrose,” Wien, 1907.

There is a good deal of presumptive evidence that the effect of phosphorus is a general one, and that the mouth symptoms are a mixed condition.

Through the courtesy of Mr. Walter Palmer I was able to examine the blood of six persons working in a match factory. There was no phosphorus necrosis or fragilitas ossium, but there appears from the table to be some diminution of the alkalinity of the serum when tested with H_2SO_4 . The experiments are too few to draw any very definite conclusion from, but the tendency seems to be a decrease of alkalinity more marked in the case of those most exposed to phosphorous fumes.

TABLE VIII.—EXAMINATION OF ALKALINITY OF BLOOD IN SIX PHOSPHORUS WORKERS.

No.	Age	Years employed	Process	Concentration of NH_4SO_4 required to just neutralise an equal volume of serum using litmus as indicator.
1	55 (M.)	17	Dipper	N 50
2	50 (F.)	30	Cutter down	N 40
3	?	20	Mixer	N 50
4	17	3	Dipper's boy	N 35
5	?	15	Dipper	N 50
6	17	3 months	Drying room	N 35
7	34 (M.) ¹	—	—	N 35

¹ Control person.

Lornisser suggested the surcharging of the blood with phosphorus as a cause of fragilitas ossium, and my observation would suggest some such association. My own impression is that minute traces of phosphorus, inhaled say during the period immediately following a meal, would tend to the combination of the phosphorus with the calcium of the bones; calcium phosphate, generally insoluble, is soluble in a mixture of acid, calcium and sodium phosphates. The onset of fragilitas ossium and the development of necrosis of the jaw is slow, and it may well be that the gradual removal of lime salts from the bones predisposes to septic infection, the mouth being the most common area to suffer from chronic septic conditions. I can advance little direct evidence in support of this view, but it seems reasonable to suppose that fragilitas ossium and jaw necrosis bear some such relation to a common cause.

DISCUSSION.

The PRESIDENT (Mr. Leonard Matheson) said the paper would add not a little to the knowledge of the subject in various important particulars, and it certainly threw a valuable light on the result of both the carrying out and the non-carrying out of the special rules in force.

Mr. F. J. BENNETT congratulated the author on the good work he had done for his fellow-countrymen by his valuable investigation. Without ignoring the valuable points the author had brought forward, the special one that interested him, and which he hoped the author would go more fully into, was the chemical pathology of the action of phosphorus in some way becoming soluble. If the author intended to go further into the matter it might be possible to consider the view that had lately come forward with regard to the action of the lipoids and fats in the isolation of germs or poisonous substances in the blood. It appeared to be a departure which promised to afford a most valuable insight into the workings of the body, and while the author had cases before him for observation, it would be very valuable if something could be learnt as to the exact way in which phosphorus withdrew the calcium from the bone. A whole evening might be devoted to that subject, and it would necessarily throw a light on the development or destruction of teeth.

Mr. W. B. PATERSON regretted that the paper had not been brought for discussion before a meeting of the British Dental Association, as in that case there might have been founded upon it some suggestions or recommendations to the Home Office, assisting to promote the Bill to which the author had referred for the abolition of the use of white phosphorus. He himself had not seen a case of phosphorus poisoning for ten years, but before the regulations came into force it was not an uncommon thing to see phosphorus poisoning at St. Bartholomew's Hospital. On the last occasion he remembered the patient came from the "boxing room" of a match factory, and he thought at the time it was very strange that a man from that department of the factory should develop phosphorus poisoning. In all such cases on inquiry it would probably be found that at some period or other of his work the patient had been in a room devoted to the dipping of matches, and on questioning the man closely he found he had been doing a little work in the dipping room, that he had had a dental extraction, and following the extraction his troubles began. Most of the cases of phosphorus poisoning he had seen had followed dental extractions, but cases of phosphorus necrosis also developed in abscessed or dead teeth, especially in the mandible. He had never seen "fragilitas ossium" in connexion with phosphorus disease of the jaw-bone. The author had brought out very clearly the absurdity of the system of appointing dentists to deal with employees in a match factory and then appointing a surgeon or a doctor to certify that their work was properly done. Certification of dental work should be by dentists, and if yellow or white phosphorus was to be still used, certification by a dental surgeon was absolutely necessary.

Mr. NORMAN BENNETT was very much interested in the reference to cases of fragilitas ossium associated or not with phosphorus necrosis and in the

suggestion of the author that instead of phosphorus necrosis being primarily an infection of the bones of the jaw through some lesion of the mouth or through the pulp chamber of a carious tooth, it might be part of a general phosphorus poisoning of which the mouth was only the chief local manifestation on account of its septic condition. If that was so, then it was to be imagined that the absorption was through the tonsils or some part of the alimentary tract or some part of the trachea or bronchi or lungs. That view, however, did not seem quite to correspond with the deductions from the improvement of the general health as a result of attention to the teeth and the condition of the mouth, and on that point he wished to have the further views of the author.

Mr. W. HERN asked whether the cases of phosphorus necrosis noticed by the author occurred in patients who had had any pyorrhœa or mischief in connexion with the gums. It had been held that phosphorus necrosis occurred chiefly after extraction and that the lesion of the extraction was the place of entry for the poisoning. It seemed to him that spongy gums might be a favourable focus for the introduction of the poison.

Mr. KENNETH GOADBY, in reply, said he should be delighted to go into the chemical pathology of phosphorus poisoning, but, fortunately, there were not many cases to be obtained, and also the amount of time allowed by the Home Office did not permit of very much time being spent on a secondary matter. With regard to the complementary deviation, that might possibly apply to phosphorus poisoning. He had tried it in the determination of changes in resistance in lead poisoning, but he could not say that it would be of great value in connexion with metallic poisoning, or poisoning by metalloids such as phosphorus. As a rule complementary deviation worked better with things of the nature of extracts. In phosphorus poisoning there was fatty degeneration of the liver, and there might be thrown out into the body a certain amount of lipoid material which would produce complementary deviation. It was impossible, however, to give any ideas on such a subject without laboratory work, and as he had not done any laboratory work he was afraid he could not answer the question any further. As a matter of fact phosphorus was naturally eliminated from the blood by the kidneys. A large number of estimations had been made from time to time to determine, if possible, some change in the quantity of phosphate discharged by the urine of persons working in phosphorus factories, but there were no satisfactory conclusions. It could not be shown that persons working in phosphorus factories were discharging more phosphorus by means of their kidneys than were normal persons, but it had been shown that persons working in phosphorus factories who were not the subjects of *fragilitas ossium* still had a diminution of the lime salts in their bones generally, not only the long bones but the jaw also. It was found by one investigator that there was about 2 per cent. of diminution of calcium in the bones. The small amount of phosphorus required to produce a slight amount of solution of calcium phosphate in the bone would not require to operate for any great length of time in the blood. With regard to Mr. Paterson's remarks, if any of the statistics in the paper were of value to the British Dental Association he should be very pleased to hand them over. The

Bill to be brought before Parliament this Session was a Bill that was agreed upon by the manufacturers themselves. It involved in one way a question of protection because no foreign-made matches containing white phosphorus would be allowed to be imported, and therefore he was not sure how such a Bill would be dealt with by the House. It was not a case for him to discuss. With regard to the inspection by dentists, Mr. Paterson would find in his report in the Blue-book that he had recommended expert dental inspection annually or biennially of the work done in phosphorus factories, because it was obviously impossible for anyone but a dental surgeon to look after the work that had been done by a dentist. He also recommended very strongly that in future the appointment of a dental surgeon to a factory should be held by a person holding a registerable dental qualification. With regard to Mr. Norman Bennett's question of the primary condition of the jaw, it was a question of pathology he should like to go into, but one too wide to attempt at the present moment. Probably the absorption went on through the lung. Recently he had made a number of experiments with regard to the absorption of lead from the lung, and he found that certain compounds of lead were easily absorbed from the internal surface of the lung, and as the breath of workers in phosphorus factories gave the examiner a taste of lucifer matches, he thought there was no doubt a large amount of phosphorus might be absorbed through the surface of the lung. It would be more reasonable to hang the two pathological conditions on one string, fragilitas ossium and phosphorus necrosis due to some generalized condition affecting the bones, than to have one due to absorption of lime salts and the other to the local effect of phosphorus. One investigator found the greatest difficulty in giving necrosis to animals even when he had fractured the teeth or the bones of the jaw. It was much more likely there was some general underlying condition that first of all lowered resistance and so allowed micro-organisms to produce infection. With regard to the dental condition, although ventilation was better than it was, no amount of efficient ventilation in any factory was ever going to protect a workman who was careless. He had seen men warming their food on the dipping-stone alongside and in the phosphorus paste, and no amount of ventilation would protect such men. Given local conditions in the mouth, plus the absorption of the fumes, then he thought phosphorus poisoning might take place through the mouth. In answer to Mr. Hern, he had occasion to report three cases of necrosis in the course of his inquiry, and all three were directly attributable to pyorrhœa alveolaris rather than to the extraction of teeth. Both Dr. Teleky in Austria-Hungary and M. Gilbert in Belgium strongly insisted on infective periodontitis as being an extremely common cause of phosphorus necrosis in factories.

The PRESIDENT (Mr. Leonard Matheson) then delivered his inaugural address.

On the motion of Mr. F. J. BENNETT, a hearty vote of thanks was unanimously accorded to the President for his eloquent and able address.

Odontological Section.

March 22, 1909.

Mr. C. F. RILOT, Vice-President of the Section, in the Chair.

A Review of the Progress of Dental Science and Literature from the Earliest Ages.

By ASHLEY DENSHAM, M.R.C.S., L.D.S.

WHEN first I was entrusted by you with the care of what was then the Library of the Odontological Society of Great Britain, I was greatly impressed by the wealth of interesting books it contains and the little use made of such valuable material. As I found myself so ignorant of the earlier history of our special branch of surgery I thought that possibly a similar ignorance amongst my fellow-members might account for the lack of interest shown in our library. I therefore set to work to glean what information I could from various works in our collection, the result of which I give you in this sketchy and incomplete review.

As a foreword I would quote some remarks of Cockayne, the learned translator of Anglo-Saxon manuscripts. "Man has an ever-recurring proneness to make himself the standard of truth, to condemn, sneer at, and despise all that he does not choose or is unable to comprehend; so in a greater degree every generation of men admires its own wisdom, skill, science, art and progress; it calls its own whatever it has learnt from men of former days, and counts the few improvements which have had their birth in its own time as triumphs and distinctions which elevate it above all the past. If we consider the history of the ages gone by, these high pretensions will soon abate somewhat of their confidence. The progress of those contrivances towards our comfort which we sum up in the term civilization has been very creeping and laborious."

The earliest references to Odontology are to be found in Egyptian literature. A large number of dental diseases and their medical treatment are mentioned in the "Ebers Papyri." These records were discovered in 1873, at Luxor, by Professor Georg Ebers, and by him translated. He describes them as a sort of medical compendium, commenced about 3700 B.C., and ending about 1550 B.C.

Of dental diseases are described gumboils, alveolar abscess, painful cervical erosion of the teeth, and inflammation of the blood of the teeth, or pulpitis. For the cure of these conditions many prescriptions are given in the form of plasters and vegetable decoctions—honey, oil, &c., being also mentioned. For the preservation and strengthening of the teeth powdered flint stones, verdigris, olibanum and honey, &c., are recommended.

An interesting light is also thrown upon the state of advancement of the parent medical profession, as an account is given of the outflow of blood from the heart with a description of some of the diseases and symptoms resulting from abnormalities of this organ. The use of the knife and cautery are mentioned for the treatment of abscesses and fleshy tumours. It is also quite evident from the anatomical descriptions given that dissection of bodies by surgeons, perhaps in conjunction with the embalmers, was a common practice. There is not recorded in the "Ebers Papyri" any form of surgical treatment for diseases of the mouth and teeth.

Herodotus, who visited Egypt long after the period already dealt with (in the year 450 B.C.), mentions the existence of dentists, but the authorities tell us that these practitioners were not able to fill teeth.

Professor Ebers in his wide researches found no trace of conservative dentistry.

Professor Emil Schmidt, of Leipzig, a noted craniologist, says that in his examination of a collection of several hundreds of mummy skulls: "I found in no single tooth anything that could be regarded as treatment by a dental surgeon. No filling, no filing or excavation of carious spots, and no artificial restoration."

Professor R. Virchow, in his examination of old Egyptian skulls, many of them royal skulls, including that of Rameses II, found no evidence of dentists' handiwork.

The frequent references made to the discoveries of mummies' teeth containing gold fillings rest upon the erroneous comprehension of the following facts, according to Dr. Geist-Jacobi. After the operation of embalming, especially amongst the defunct of the upper classes, the

eyebrows, the tip of the nose, the lips and the teeth of the corpse were gilded, and a gold coin placed between the teeth, or the tongue was covered with thin gold plate. The gold, however, had nothing to do with the conservative treatment, or restoration, of the teeth.

As to whether mechanical dentistry was practised by the Egyptian dentists of those times it is more difficult to speak with certainty. Professor Ebers says that as the Etruscans knew the art—as shown by a specimen in the museum at Orvieto, taken from an Etruscan tomb dating from the fourth to the sixth century B.C.—it is most probable that the Egyptians were not ignorant of it. However, this view does not seem to be borne out by the examination made of the skulls of the wealthy classes.

Amongst that other ancient race the Hebrews, of whom we have detailed history, we find no reference to the prevalence of dental diseases. In those excellent sanitary laws drawn up by Moses for safeguarding the health of the Hebrews under his care during their wanderings from Egypt to the Promised Land no rules are laid down for the care of the mouth and teeth. This is probably because the Hebrews rejoiced in sound and healthy teeth. There is no evidence for supposing that the omission in this respect was due to carelessness or lack of appreciation of the teeth. On the contrary, the teeth were evidently regarded as objects of beauty and great value, for in the criminal code drawn up by Moses it is enacted that if any man do injury to another then shall he give life for a life, an eye for an eye, and a tooth for a tooth. And it is further enacted that if any man knock out his slave's eye or tooth, then must he let that slave go free for the sake of his lost eye or tooth.

Amongst the Greeks, during the first centuries of their civilization, we find no reference to dentistry or other sciences, the writings up to the time of the Siege of Troy, supposed to be about 1184 B.C., being almost entirely hymns in praise of the gods. Homer, in his *Iliad*, written about 900 B.C., does not mention the subject, although he frequently describes how the javelin or the sword crashed through the teeth of the warriors; from which it would seem that the services of a dentist were often needed, though not forthcoming. A few centuries later Herodotus and Aristotle give us some information on the subject of the teeth, but as Aristotle tells us that the instruments of the tooth-doctors are the scraper and the extractor, we gather that dental operations then were very restricted in scope, and included only the extraction of loose teeth and the removal of deposits of salivary calculus.

Hippocrates, born about 460 B.C., devotes several chapters to the description and treatment of dental and oral diseases. He recommends the extraction of very loose teeth with leaden forceps, but regards the extraction of firmly fixed teeth as a dangerous operation. He describes the troubles sometimes attending the eruption of wisdom teeth, also alveolar abscess and necrosis of the jaw-bones; but the treatment recommended does not extend beyond the use of fomentations and mouth washes. He describes the period of the first dentition, and draws attention to the many ailments that afflict infants at this time. He gives detailed directions for the treatment of fracture of the mandible by means of a leather chin splint and bandages, a method which survived for two thousand years. Ulceration of the tongue due to sharp edges of decayed and broken teeth is noticed by him; and to him was ascribed by his later commentator Galen the first use of the file for smoothing and shortening teeth. After the subjugation of the Greeks by the Romans about 160 B.C., their history becomes merged in that of the Roman Empire.

Amongst the Romans we have definite documentary evidence that dentistry was extensively practised at an early date, for one of the laws of the twelve tables passed in 450 B.C. refers to teeth sustained by gold wires. Here it is made unlawful to bury jewels with a corpse, exception only being made in the case of teeth bound together with gold wires. There are to be seen in the Civil Museum of Orvieto in Italy two small dentures composed of soft gold bands carrying teeth apparently carved from ivory. These were removed from an Etruscan tomb and date from the fourth or fifth century before the Christian Era.

Cornelius Celsus, born 25-30 B.C., writes very fully on the subject of dentistry, one very interesting description he gives being that of the proper method of extracting a tooth. He says the gum must first be scraped away and separated from the tooth until the latter is quite loose, as it is very dangerous to extract a firm tooth. When it is possible take out the tooth with the fingers, or if this is not possible use forceps. If there is a large cavity in the tooth, fill it with lead or lint to prevent fracture. Here is the first mention of filling carious cavities in the teeth, not, however, for their preservation.

Celsus recommends treatment of a fractured mandible by tying together the teeth on either side of the seat of fracture with silken ligatures; he also advocates the use of the splint described by Hippocrates.

In the year 50 A.D. Scribonius Largus, in the reign of the Emperor Claudius, propounded a new theory of the causation of dental caries,

ascribing it to the action of small worms, and for their destruction he prescribed fumigation. This idea of the causation of dental caries existed until the middle of the eighteenth century, and all our modern scientific researches have but succeeded in reducing these worms to a microscopic size.

About 100 A.D. Archigenus, surgeon in the time of the Emperor Trajan, discovered and described the operation of rhizodontrophy for alleviating the pain arising from inflamed and discoloured teeth.

Pliny the elder, historian and proconsul, born in 23 A.D., was in his later years a prolific author, thirty-seven volumes of his writings being extant; he is described, by the way, as having fallen a victim to his desire for knowledge in an eruption of Vesuvius 79 A.D. He emphasizes the necessity of thoroughly washing the teeth with salt, and the use of an uneven number of mouthfuls of water (1-3-5, &c.), and advocates the use of tooth-picks to prevent bad odours in the breath. He affirms that there is a poison in the human teeth which dulls the lustre of a mirror and is fatal to the unfledged young of doves. He gives far less practical advice for further prophylaxis in that he advises everyone to eat a mouse every two months to ward off pains from the teeth. He gives many prescriptions for tinctures and tooth powders, deprecates extraction of teeth, advocates filing elongated teeth in two or more sittings, and divides dental pain into three groups—the first having origin in the teeth, the second in the gums, and the third in compression of the nerve.

Loosening and early loss of the teeth seems to have been very prevalent amongst the Romans, as all the authors devote attention to it and give many prescriptions for its prevention and cure; most of the ingredients of these being very unpleasant, such as the powdered excrement of the mouse, &c. The mechanical branch of dentistry was extensively practised in the Roman Empire by slaves and freed men. It was crude, and consisted in joining together several teeth that had come out, and later teeth carved from ivory and other animal teeth, and attaching them to the adjoining teeth with strips of soft gold, horsehair and silk ligatures. This work commanded high fees and led to specialization in dentistry, as the doctors of the period did not concern themselves with any but medicinal treatment.

The name of one Casellius has come down to us as that of the first dental practitioner. He had a shop on the Aventine Hill in Rome during the reign of the Emperor Domitian about 90 A.D. Martial tells us that he was renowned throughout the Empire, and became as rich as a senator; also that he was held in great esteem by ladies of high degree,

in that he was able to restore by means of his art the charms which they had lost.

Whatever progress was made in the dental art during the Empire seems to have been lost after its fall, as the only writer of later times who devotes much attention to the teeth and their maladies was Galen, born in 131 A.D., and although he wrote fully on the subject he tells us nothing new. Other medical writers for several centuries devote only very slight notice to the teeth, and we find no references in the literature of the times to dentists practising their art.

A few references to Latin authors of the following two or three centuries will show how little advance was made during that period.

Adamantius of Alexandria, in the end of the third or beginning of the fourth century, draws attention to the influence of the canine teeth on the shape of the face and draws deductions, from their relative positions, as to the character of the individual.

Marcellus Empyricus, at the commencement of the fifth century, gives the following advice for extracting painful teeth. Anoint the patient's nose with the marrow of a hare and green oil, tell him to hold his breath and clench a stone between his teeth and then yawn widely. At this moment the humour producing the pain flows out and the tooth can easily be removed without causing suffering.

Aetius of Amida, in the middle of the sixth century, claims the discovery of the use of the file for elongated teeth. He elaborates Galen's theory of nerve-supply of the teeth and says that fine terminal branches of the trigeminal nerve enter through the open ends of the roots of the teeth, and for this reason they only, of all the bones, are sensible of pain.

Here we may leave the consideration of dentistry in antiquity and pass on to the so-called Middle Ages. In these times, following the decline and fall of the Roman Empire, civilization, with its attendant arts and sciences, is found most highly developed in Arabia and the North of Africa, and it is in the writings of the Arabian savants that the history of dentistry for several succeeding centuries is preserved to us.

Abou Bekre Mohammed ben Zakaria el Razi, later commonly referred to as Rhazés, was born in Persia in 850 A.D. He counsels the avoidance of extraction of teeth. For periodontal inflammation he recommends scarification, the use of opium, and the application of leeches. Carious cavities, he says, should be filled with a mixture of alum and mastic. He records a case of re-formation of a mandible after total destruction by necrosis.

Later on Avicenna (980-1037) wrote as his chief work the "Canon"—a treatise on pathology and therapeutics. In the chapters dealing with the teeth he describes the acute pulsating pain felt in a tooth as due to the root containing too much fluid, and advises perforating the root to let out the fluid and permit of the application of remedies. He gives the following directions for the avoidance of dental troubles: Do not eat substances likely to decompose—such as fish and milky foods; avoid things very hot or very cold—especially in rapid succession; do not eat hard substances, and do not pick your teeth. Rub them frequently with honey or baked salt.

We find that these and other Arabian writers of the times have derived all their knowledge from, and based their treatment upon, the older Greek physicians, and beyond further elaboration of their already complex prescriptions have not really advanced the science of dentistry in any respect. It is evident that the profession confined itself entirely to the surgical aspect of dental disorders, and left the mechanical side of prosthetic dentistry to quacks and charlatans. So much so that Abulcasis thought it necessary to warn the public against those ignorant persons who confined their efforts to the manufacture of artificial teeth. "Al-tasrif," the *magnum opus* of Abulcasis, otherwise called Absarabius, is interesting and shows some originality of thought. In it he describes his treatment of dental fistulæ with the button cautery. He depicts there, too, a large number of dental instruments. He writes at length on the subject of tartar and the necessity for its removal, and devised a set of instruments for this purpose. The method of extraction he advocates would not find favour with our modern patients. He advises, first, that you find the right tooth, not necessarily the one the patient points out; then cut away all round with a scalpel; then take hold of the tooth with light forceps having short handles, and gently move it to and fro. Then take the patient's head between your knees, and with stronger forceps having steel handles and corrugated blades use force sufficient to remove the tooth, taking great care not to break it; for, he says, a root left behind often gives rise to greater pain than the original. He describes the use of files and enamel chisels for separating teeth, also how to ligature loose teeth with gold wires, and how to make and fix a substitute for a lost tooth, carving it from ox-bone. He also describes salivary calculi, and how to remove them from the sublingual duct.

The decadence of Arab influence in civilization coincides with the period of the expulsion of the Moors from Europe. Through many

succeeding centuries, however, their methods were practised and their *nostra* administered, in the treatment of dental disorders, by the hordes of quacks, mostly of Jewish origin, who wandered throughout the length and breadth of our continent.

We might here take a glance at the condition of dentistry in our own country. The ancient Britons living in natural uncivilized conditions—dressed chiefly, as we understand, in their birthday dress stained with woad—presumably had no dental disorders other than traumatic ones, and what treatment may have been required was, no doubt, supplied by the Druids. Dental surgery must, therefore, have been imported into our isles by the Romans during their occupation, which began about A.D. 55. There is every reason to believe that the art was practised here throughout the 400 years this lasted, for, although I find no record of dental instruments having been found amongst Roman remains in Britain, large numbers of dental forceps have been recently found amongst late Roman remains at Homburg in Germany, and what was practised in one Roman colony was most probably practised in others. About 411 the Roman legions were recalled to Italy to repel the invading Goths, and when England became England about 449 by the incursion of the Angles and the Saxons, the immediate progenitors of our English race, these latter proceeded to exterminate the ancient Britons, and with them all trace of Roman civilization. During the 550 years of the Anglo-Saxon period the treatment of diseases by herbal decoctions, charms and incantations prevailed, even whilst knowledge of the Hellenic School flourished amongst the Arabs. In the "Master of the Rolls" series of records there is a volume entitled "Leechdoms, Wort Cuning and Starcraft of Early England," translated from the collection of Anglo-Saxon manuscripts, and edited by Cockayne.

In this, Marcellus, A.D. 380, directs, as a cure for toothache, to say "Argidam, margidam, sturgidam!" also to spit in a frog's mouth and request him to make off with the toothache. In the *Herbarium of Apuleius* there are 185 herbal remedies for various disorders, including many for toothache, of which the following are samples: "For the toothache take the same wort betony and boil it down in old wine or in vinegar to the third part; it will wonderfully heal the soreness of the teeth and swelling. For sore of the teeth, and if they wag, take the wort *Britannica*, it out of some wonderlike virtue will help. For toothache give to eat heads of the *solsequiam*. For toothache take a root of *Bothen*, give it to eat, without delay it removes the sore of the

teeth, and let him hold the ooze in his mouth, soon it healeth the teeth. Leaves of this same wort, bone-wort, bruised and mingled with honey, heal the canker of the teeth, from which often the teeth fall out. For sore of the teeth and gums take seed of the same wort, lousebane, seethe it in vinegar; let him hold then in his mouth some of the vinegar for a long while; sore of the teeth and of the gums and all rottenness of the mouth shall be leechened."

The next manuscript of interest is Bald's "Leech-book." This was written about 900 A.D., and probably at some time belonged to the Abbey of Glastonbury. According to an inscription on it, it was originally the property or even the work of one Bald, a leech or doctor. In section 6 of the first book are given seven "leechdoms for sharp pain in the teeth, either for the upper toothache or for the nether"; of these I will quote only one. "For tooth wark, if a worm eat the tooth take an old holly leaf, and one of the lower umbels of hart-wort and the upward part of sage; boil two doles in water, pour into a bowl and yawn over it, then the worms shall fall into the bowl. If a worm eat the teeth take holly rind over a year old and root of carline thistle, boil in so hot water, hold in the mouth so hot as thou hottest may. For tooth worms take acorn meal and henbane seed, and wax of all equally much, mingle these together, work into a wax candle and burn it, let it reek into the mouth, put a black cloth under, then will the worms fall on it." In section 4 of book iii. it says: "For toothache chew pepper frequently with the teeth, it will soon be well with them again, seethe henbane roots in strong vinegar or in wine, set this into the sore tooth and at whiles chew with the sore tooth, it will be well. If the teeth be hollow chew rosemary roots with vinegar on that part."

In a manuscript of this time concerning schools of medicine one section deals with "the flesh which waxeth about the teeth, and maketh the teeth wag, and disturbeth them." Another section deals with the nature of teeth as described by the Greeks, and of the causes of toothache it says: "Often the worst humour cometh to the teeth from the head in such manner as it droppeth off a house upon a stone, then it getteth the better and drilleth through and pierceth the stone, similarly the moisture of the head from above falleth upon the teeth and pierceth through them and causeth them to rot and swell, so that the teeth can endure neither heat nor cold, especially the grinders teeth which are fastened each with four roots, and then they leave their roots, then they turn swart and fall." From the relatively numerous remedies given for

dental disorders in these manuscripts, it is evident that our Anglo-Saxon forefathers suffered considerably from dental disease.

For the first three centuries after the Norman conquest of England in 1066 there are practically no records of social life; and as we are dependent upon Chaucer and the poem of *Piers the Plowman* for any knowledge of the intimate details of the habits of the various grades of society in those times, we can guess from what they tell us that the serfs and poorer classes found great difficulty in sustaining life at all; the merchant classes had enough to occupy them in extending their trade and paying their taxes, and the upper classes were continually occupied in fighting the French, Scotch and Welsh, and in fighting amongst themselves. The arts and sciences therefore which need prosperity and security in national life for their development must have been in an embryonic or non-existent state in England in the Middle Ages.

Probably in England, as on the Continent, wandering empirics of Jewish nationality who had obtained their methods from Arabian sources practised dentistry in these times; but as the Jews were expelled from Britain in 1290, and Green the historian tells us that from that time up to the time of Cromwell no Jew touched English ground, even this degree of skilled attention was lost to us. Force must have been the chief factor in tooth extraction in those days, as I have discovered a copy of a fourteenth century manuscript illumination in which a tooth-drawer is depicted at work. He has round his neck a wreath of past trophies in the form of a string of apparently sound molars. He is using an enormous pair of forceps seemingly 2 ft. long, and the handles are held each in one hand after the manner of modern grass-cutting shears.

As the power of the Roman Catholic Church increased, and monastic orders were established, the monasteries became the centres of surgical and medical practice—very empiric in quality and lacking in progress—whilst our branch received there very little attention. In 1160 the clergy were forbidden by the Church authorities to practise the surgical part of medicine as something low and unworthy, and on the ground that the Church abhors blood. The members of the monastic orders deputed to undertake the work of barbers and shavers of tonsures for the monks had gradually assumed the duties of minor surgery and tooth extraction, and in the course of time many of these separated themselves from the monasteries, and, practising entirely among the laity, established the profession of barber-surgeons. A certain amount of light is thrown upon the condition of dentistry in these times by the

recorded history of the Barbers' Company. A charter was granted to the barber-surgeons by Edward IV. in 1462. It begins by reciting how "Our beloved, honest and free men of the trade of barbers of the City of London, using the craft or faculty of surgeons, have for a long while diligently and laboriously occupied themselves with the wounds, bruises, hurts and ailments of our lieges, as well as in bleeding them and drawing their teeth."

In the course of time dental operations were more and more neglected by the barber-surgeons, no doubt owing to the difficulty and uncertain success attending the operation of tooth extraction with crude instruments and no knowledge of dental anatomy, and the loss of prestige to a settled practitioner consequent upon frequent failures in this operation; so this department fell again into the hands of the peripatetic quacks, who were careful to leave the district before the painful results of their failures became apparent. In the reign of Henry VIII., in 1540, an Act of Parliament was passed uniting and incorporating the separate bodies of surgeons and barber-surgeons. This Act, whilst confirming the privileges granted in the charter of Edward IV., and conferring several new ones, includes a provision that "no manner of person within the City of London, suburbs, and one mile therefrom using any barbery, shall occupy any surgery, letting of blood or any other thing belonging to surgery, except drawing of teeth," &c., &c. Thus was the practice of tooth extraction, as it were by law, relegated to the shavers and charlatans. There were, however, apparently still some dentists in those times who conducted their practices on professional lines, and were held in some esteem by their surgical colleagues, for there is an entry in the records of the Barbers' Company in 1551: "John Bryckett, tooth-drawer, hath been admitted for a brother in this house."

(Since writing this paper my attention has been called to a series of articles on the early history of dentistry in England which have appeared in the *British Dental Journal*, and to these I would refer you for a fuller account of this period.)

To return from this chronological digression. Amongst the Germanic races in Europe in the earlier centuries of the Middle Ages the healing art was entirely unknown, and dental troubles were treated by incantations and the use of substances having supposed magic properties. The favourite method, for instance, of encouraging the painless eruption of children's teeth was to suspend round the neck of a child a thread soaked in blood by passing it through the eyes of a living mouse.

The foundation of the earlier universities, which succeeded the older monastic colleges of Salerno and Monte Cassino, did not much improve matters in this respect. We do, however, find amongst the writings of several of the Italian professors of medicine and surgery some scanty descriptions of dental diseases and their treatment. Of these writers may be mentioned:—

Bruno de Longoburgo of Bologna (1252).

Lanfranchi (1300), who recommends the use of narcotics for alleviating the pain of toothache.

Guy de Chauliac, a celebrated surgeon in the latter half of the fourteenth century, gives merely a *réchauffé* of the ideas of Abulcasis on the teeth. He was in some other respects a reactionary in that he taught, on the lines handed down from Galen, that the promotion of suppuration was necessary for the healing of wounds, and so by his great authority and weight stopped the progress that might have been made in promulgating the theory of his brilliant predecessor Theodoric that suppuration was not only unnecessary to the healing of wounds, but actually retarded the process.

Pietro di Argelata of Bologna (1452) writes on similar lines, and also recommends the use of strong acids for cleansing carious cavities in teeth.

In 1470 Johannes Platearius, professor at Pisa, recommends the sitting posture as the most convenient for the patient during an operation; evidently an innovation. He also lays great stress upon the necessity for plenty of fresh air during the operation of extraction, seeming to have a prophetic knowledge of later theories as to air-borne infection of wounds.

The last writer of this period to be mentioned is worthy of particular note. Giovanni d'Arcoli, professor at Bologna and Padua, died in 1484. He wrote at length and with originality on the subject of the teeth. He recommends the use of thin gold leaf for filling carious cavities, having first cleansed the cavity with strong acids. This is the greatest advance so far towards conservative dentistry, no more durable substance having been, up to this time, employed for this purpose than mastic and wax.

The worm-theory of caries receives full recognition at the hands of all these later authors.

SIXTEENTH CENTURY.

In the sixteenth century we find a greater number of authors on dental subjects, and amongst these may be mentioned Alessandro

Benedetti, professor at Padua, and Giovanni da Vigo, surgeon to Pope Julius II; he deplores that the extraction of teeth should be in the hands of quacks, and writes of gold fillings. However, he relegates other and more serious surgical operations, such as the radical cure of hernia, treatment of cataract and vesical calculus, to the "vagabonds qui courent de pais en pais."

The first treatise on the teeth appearing originally in a language other than Latin or Greek was written in 1572 by Walter Ryff in German. It deals with the face, eyes and teeth, and in it he gives direction how to keep the mouth and gums clean, healthy and in good condition. He describes and depicts many instruments, scalers, forceps and keys. This work was soon followed by a brochure in French published in 1582 by Urbain Hémard, surgeon to Cardinal Georges d'Armagnac. It was entitled, "A Study of the True Anatomy of the Teeth, their Nature and Properties, and the Diseases to which they are Subject."

I will not weary you by referring to all the authors of this period, but one stands pre-eminent. Ambroise Paré was born about 1510, and died in 1590. His writings on surgery are numerous, and their importance and influence can be deduced from the fact that the eleventh edition appeared in 1652, 100 years after their original publication. He describes the macroscopic anatomy of the teeth, their attachment to the jaws, and their sensitiveness. He advises the use of hot garlic and oil of cloves for the alleviation of toothache, and recommends the use of vitriol and the actual cautery for the destruction of the caries-producing worms. In Paré's works we find the first mention of transplantation and replantation. He apparently did not himself practise these operations, but enumerates cases. He does not write at length upon mechanical dentistry, but indicates that in his time artificial teeth were made of carved bone and ivory, and were fixed to the adjoining teeth with gold and silver wire. The use of obturators is described by Paré, and most probably they were introduced by him. The obturator was made of a thin gold or silver plate, somewhat larger than the orifice to be covered, to the convex surface of which a sponge was fixed; this sponge, lying in the nasal cavity, absorbed moisture and so swelled, in this way holding the obturator in position. He also describes artificial noses made of gold or silver plate, or of cardboard; these were covered with tinted linen and held in position by a cord passed round the back of the head. Paré also recommends deep incisions into the gum over erupting teeth.

Of other writers Peter van Foreest, professor at Leyden, recorded many useful clinical observations about 1580. He condemns the consumption of sweetmeats as harmful to the teeth. He also describes many cases of inflammation and extensive tumours resulting from the presence of artificial teeth, and on these grounds condemns the use of artificial teeth *in toto*.

Theodore Zwinger, a Swiss professor about this time, was a timid man. He advises that in order to avoid responsibility for the grave accidents which frequently attend the operation of extraction of the teeth these operations should be left to the quack tooth-pullers.

An amusing and interesting case which received a good deal of notice in the literature of this period was that of the boy of Silesia with the golden tooth. It is recorded by Jacob Horst, professor of medicine in Helmstadt, that there was in 1595 at Schweidnitz in Silesia a boy aged 10 who had a golden tooth. Horst proceeded to write a book about it: "*De aureo dente maxillari pueri Silesii*." He describes this as a supernatural event due to the birth of the boy taking place on December 22 when the sun was in conjunction with Saturn in the zodiacal sign of Aries, and deduced from this that the time of the golden age was at hand. The deductions and predictions published in this book were widely criticized by Horst's colleagues. In particular, a Scotchman, Duncan Liddell, heaped ridicule on Horst's ideas, and stated that if the sun were in conjunction with Saturn in December he should consider it a much more wonderful event than for a boy to be entirely made of golden teeth, seeing that the sun does not enter the sign of Aries until March. He also pointed out that the gold on the tooth stopped short of the roots.

Another doctor, Balthasar Camindus, records that for many months before the date of his writing the guardians of the boy had refused to allow him any longer to be examined by experts, from which he deduced that the tooth was only covered by gold leaf and that the roots of the tooth were beginning to show their base composition owing to further eruption of the tooth. It appears that the tooth in question was the first lower molar on the left side, and that the second premolar was missing, probably not having erupted, the second temporary molar having been shed. The golden tooth would seem to our prosaic minds to have been the work of some clever jester, born before his time, who was tempted by the ease of access to the tooth to create and exploit a lucrative freak. He may have overlaid the tooth with gold leaf, or have adapted a gold crown. If it was a gold crown it is certainly the first recorded instance

of this common object of latter-day prosthetic invention. An explanation advanced by a German critic is that the tooth was covered by golden yellow tartar, but this is surely to credit the observers of that time with a very slight acquaintance with the desirable metal, if they could mistake lime salts for gold.

The seventeenth century was not marked by any great discoveries or advance in odontology. Forty or more books and pamphlets of German authorship were published during the century. They mostly dealt with toothache and the ailments of the dentition period, and we do not find in them anything particularly worthy of note. In France during this period the dental quacks flourished. The Pont Neuf was the centre of their activity in Paris. They were not over modest in their advertising methods, and appear to have had a somewhat flamboyant taste in costume, leaning towards richly embroidered cloaks and jewelled turbans as their most suitable working dress. Some of them are reported to have amassed large fortunes and to have had hankerings after political power. It is from the lampoonists of the time that the names come down to us of Arnaut, Carmeline, Brioché and le grand Thomas, as gentlemen of this persuasion.

Dupont and Pomard, Frenchmen, about 1633, recommended the extraction and replantation of painful teeth.

Mathias Purmann, a doctor of Breslau (1648-1721), put on record the most important advance of the century; he is the first to describe the method of taking an impression of the mouth in wax as a preliminary to making an artificial denture. Possibly he was the discoverer of this useful practice.

The three Englishmen of this century who are most noteworthy in connexion with our speciality are Highmore, Drake, and Cowper. Nathaniel Highmore, a surgeon practising in Dorsetshire, described the maxillary sinus called after him the antrum of Highmore and its proneness to pathological changes. It was not, however, until fifty years later that surgical operations on this cavity were performed by James Drake and William Cowper; the latter established the practice of extracting the first molar and opening the antrum through its socket. He was the anatomist who discovered the glands named after him.

The two men of the century who were the founders of dental histological anatomy are worthy of special notice and remembrance. They are Marcello Malpighi, professor at Bologna, the describer of the rete Malpighii; and Anton van Leeuwenhoek, a naturalist of Delft. The

latter was the first to construct a large microscope, and he gave a minute description of the dentinal tubules before the Royal Society here in London in 1678.

The only English book on dentistry of which we can find any mention is one by a nameless author published in 1687 and entitled "Curious Observations on that part of Chirurgery relating to the Teeth."

EIGHTEENTH CENTURY.

Here we find France to the fore in the progress of dental science. France was the first country to recognize dentistry as a distinct, specialized branch of medical science. In May, 1699, after two or three centuries of internecine war between physicians, surgeons and barbers, an edict was passed creating, as a distinct subdivision of the surgeons' guild, the surgeon-dentists, or experts.

The state of dental literature at this period is well described by the great Fauchard in the preface to his first work published in 1728. He says: "Although surgery in general has been greatly perfected in these latter times, and many important discoveries in anatomy and methods of operation have been made, and many wise and curious observations have been recorded, yet dentists do not find sufficient records to aid them in performing all their operations. If some writers have described the teeth and their special maladies, such as Urbain Hémar and Bernard Martin, they have not done so in sufficiently detailed fashion.

"There are not either any public or private courses of instruction in surgery in which the maladies of the teeth are fully taught, or where the foundations for practising the art can be acquired.

"The most renowned surgeons having abandoned this branch of their art, or at least having only very slightly cultivated it, it has, as a result, fallen into the hands of people without knowledge or experience, who practise it to the public danger without method or principles.

"It is only since 1700 that the city of Paris has opened her eyes to this abuse. Those who desire to become dentists must now submit themselves to examination. But although the examiners are without doubt very learned in all other branches of surgery, I consider, if I may be permitted to say so, that practising only those branches it would not be undesirable for these occasions to employ a dentist of known worth and experience, who would be able to test candidates as to their

knowledge of those difficulties which long experience has taught him to expect in the practice of his art."

Pierre Fauchard, born in Paris in 1690, was the first to write a book on purely dental subjects, with the avowed intention of putting all he knew about odontology at the service of those who might desire to follow him in the study and practice of his calling. His chief work, "The Surgeon-dentist, or Treatise on the Teeth," first published in 1728, is a curious medley of scientific ideas and mediæval survivals of treatment. He describes the macroscopic anatomy of the teeth on the lines of his predecessors, giving, however, a more detailed account. He also describes the microscopic structure of enamel. He devotes a good deal of attention to the health of the teeth and gives good advice as to their preservation. He condemns sugar as harmful, and considers the moderate use of tobacco as harmless. A long chapter is devoted to powders, opiates and mouth-washes, but the prescriptions are mostly bad according to our ideas. In the chapter entitled "General causes of the maladies relating to the teeth," we find only our old friends the vapours and humours in evidence. He divides caries into two classes, soft or putrefactive, and dry caries. He records and expresses his astonishment at it, that a tooth well filled is frequently cured of caries for a long time. He gives no good explanation of hypoplasia, but compares it to the rusting of iron. He denies the existence of worms in dental caries, and says that if worms are ever found they are introduced in the form of eggs with food, and doubts the presence of micro-organisms in the soft deposit covering badly kept teeth as described by N. Andry of the Faculty of Paris in 1700. In Chapter X he describes how to enlarge the openings into pulp-chambers and narrow pulp-canals by means of a bow and drill, and so foreshadows the modern dental engine. When writing on the extraction of teeth he describes the interesting methods of the charlatans of that time. These latter kept ready prepared small membranous envelopes each containing some chickens' blood and a tooth. One of these was introduced unperceived into the patient's mouth, a pretence was then made of touching the aching tooth with a straw, or the point of a sword, the assistant then rang a bell loudly, and the patient spat out blood and tooth before the eyes of the astonished and credulous onlookers. Fauchard practised transplantation, replantation, immediate torsion by means of forceps, and immediate regulation by means of the key and forceps. For filling teeth Fauchard preferred lead or tin, beaten out thin and cut into fragments of varying sizes and placed in the cavity, much in the way we use non-cohesive gold cylinders,

and there condensed and burnished. He considered that gold could not be so well adapted to the sides of the cavity and also considered its use an unnecessary extravagance. He is the first to give any clear and detailed account of the methods employed in mechanical dentistry. The materials used were human teeth, hippopotamus teeth, ox bones and teeth, and walrus tusks. He apparently did not know the use of wax modelling as already referred to, but obtained what accurate adjustment was possible by means of compasses, paper patterns and frequent trying in the mouth. He relates his invention of a method for retaining an upper denture in an edentulous mouth by means of steel springs reinforced with whalebone, attached to an armature in the mandible. He also mentions spiral springs for the same purpose, but does not recommend them. It must be noted, too, that he was the first to try enamelled teeth. He made dentures of hippopotamus ivory, to the front of which were attached strips of metal, upon which the forms of teeth had been enamelled. This in 1756. In the second edition of his book published in 1746 he mentions the application of the principle of suction for the retention of upper dentures.

I have dwelt at such length upon the work of Fauchard because his book marks a distinct epoch in the progress of dentistry, and I think he may be truly regarded as the father of dental science in that he first advocated special education in dentistry, and did what he could to advance this by publishing his methods and experiences for the benefit of others. The success of his works produced an abundant crop of publications in France during the remainder of the century, but it must be admitted that a large number of these appear to have been intended to attract the attention of the public, and not of the profession.

Jourdain and Bourdet both wrote many thoughtful books on dental subjects. Bourdet pointed out the cohesive properties of gold-foil and the use to be made therefrom.

To conclude this survey of the century in France a rapid glance may be taken at the history of the introduction of mineral teeth. In 1774 a chemist named Duchâteau, living near Paris, found the colour and odour of his ivory denture so unpleasant that he conceived the idea of having it modelled in porcelain. He applied to M. Guerrard, a porcelain manufacturer. The first experiment failed owing to the shrinkage of the denture. Then followed a number of experiments, none very successful. He then associated with himself Nicholas Dubois de Chémant, a Parisian dentist, and they made prolonged experiments with the *pâte tendre* form

of porcelain just then introduced at Saint-Cloud. They modified this considerably to reduce its fusing point, and produced a workable porcelain body, and Duchâteau communicated his discovery to the Royal Academy of Surgery in Paris in 1786. He had not, however, sufficient knowledge of dentistry to make his dentures satisfactory.

Dubois de Chémant then severed his connexion with Duchâteau and pursued his experiments alone. He altered and improved the formula and made many dentures of what he termed mineral paste, which were successfully worn. In 1790 he received from Louis XVI. a patent of invention and so deprived Duchâteau of any rights he may have had in the discovery. He then came to London, and obtained a patent for his mineral paste for fourteen years. Messrs. Claudius Ash and Sons took up the matter with de Chémant, and in 1837 were able to supply mineral teeth in quality and appearance somewhat approximating to the present perfected form.

In 1808 Fonzi, another Parisian dentist, conceived the idea of incorporating platinum pins in the mineral body, and discovered also how to impart translucency to the teeth.

De Chémant practised for many years in London, apparently with very satisfactory results to his pockets, as in one of his books he speaks of having provided dentures for more than 12,000 persons. His books, however, have no scientific value and are devoted to self-laudation and advertisement of his success in the use of his discovery. Many of the cases he cites as benefiting by his treatment are incidentally interesting, showing as they do what horrible conditions of septic absorption and fetid exhalations resulted from the bone and ivory dentures in use before the introduction of mineral teeth and metallic bases. Whatever may have been the true facts of the discovery, it is beyond question that by his life's work in this direction Nicholas Dubois de Chémant conferred a great boon upon humanity in abolishing the abominable, inartistic and insanitary bone dentures.

In surveying English dental literature during the eighteenth century we find very little advance in the technique of dental surgery; indeed, we find our English dentists a long way behind their French confrères and apparently not conversant with Fauchard's writings, but we find at home more decided and solid progress in what may be regarded as the scientific basis of our speciality.

With the work of John Hunter, dentistry finally emerged from its empirical stage and henceforward develops on scientific lines grounded upon the anatomical and physiological data given to us by him in his

"Natural History of the Human Teeth" and "Practical Treatise on the Diseases of the Teeth," published in 1771 and 1778. These books should be read by everyone interested in the study of dentistry. They are admirable in their profound thoughtfulness and lucidity of explanation. The mention of a few points dealt with by him will serve. He tells us that caries of the teeth is a disease whose nature is inexplicable, in that it appears to be peculiar to these organs and is not due to external irritation or to a chemical process; it is rarely found in the roots of the teeth, and hardly ever occurs after the age of 50. He denies that one tooth infects another. He recommends lead for filling cavities in teeth. He states that invasion of the pulp by caries necessitates its destruction by the actual cautery, and he is the first to enunciate that in such cases total extirpation of the pulp down to the apices of the roots is essential to successful filling and retention of a tooth. He describes two forms of pyorrhœa alveolaris, with and without suppuration, and considers it a disease of the alveolar periosteum. The extraction of temporary teeth as a corrective measure in overcrowding he strongly deprecates. The regulation of teeth, he says, should not be commenced before the eruption of the bicuspid, and the malplaced teeth should be brought into place by pressure. He recommends the extraction of bicuspid as a cure for superior protrusion. Benjamin Bell, of Edinburgh, in his "System of Surgery," contributed a number of valuable and important observations on dental subjects.

We must not pass without notice the names of Joseph Priestley, Professor of Chemistry at Birmingham (1733-1804), and Humphrey Davy (1778-1829), the discoverers of nitrous oxide, although it was left to others to apply its use for anæsthetic purposes.

Germany lagged somewhat behind in the eighteenth century, and of its authors we will only note Philip Pfaff (1756), who was apparently the first to make plaster models from wax impressions; he also practised capping exposed pulps; and Friedrich Hirsch (1796), who writes of a paste he had discovered for filling teeth, which hardened *in situ*, the forerunner, perhaps, of the cements of the present day. Documentary evidence as to the nature of this paste is, however, wanting.

NINETEENTH CENTURY.

At the commencement of the century we find a new country to the fore—the United States of North America. Building upon the solid scientific foundations laid by Hunter and Bell, our American colleagues

—with ingenuity and inventiveness so characteristic of their young nation—made rapid progress in the technique of our art, although their output of literature was not great.

The introduction of dental surgery into the United States was due to some French surgeons, with a knowledge of dentistry, who were attached to the troops under Rochambeau and Lafayette which took part in the War of Independence. Of these the most distinguished was Joseph Lemaire, who not only practised as a dental surgeon, but also instructed many others in his art. James Gardette, who practised in Philadelphia from 1784-1829, was another of the French pioneers of dentistry in America. Horace Hayden, in 1825, gave demonstrations in operative dentistry. Edward Hudson, an Irishman, domiciled in Philadelphia in 1805, was the first to fill root-canals with gold, and he must have found that this required a large expenditure of time and patience.

In his book, "Guide to Sound Teeth," Shearjashub Spooner claims for his brother, John R. Spooner, of Montreal, the discovery of the use of arsenious acid for the devitalization of tooth-pulps.

A notable figure in the development of our science in America was Chapin A. Harris, of Baltimore. As a young surgeon his attention was attracted to dentistry by the works of Hunter, Fox, and Delabarre. The publication of his "Principles and Practice of Dental Surgery" in 1839 marks a further distinct epoch in the progress of dental education. He was, too, chiefly concerned in the foundation of the first school of dentistry—the College of Dental Surgery established in Baltimore in 1839. He and Hayden were on the staff of that school. In the year 1840, in collaboration with Eleazar Parmly, S. Brown and E. Baker, he produced the first number of the first dental journal, the *American Journal of Dental Science*.

In 1838 Merritt, of Pittsburg, described the advantages of using a mallet for condensing gold.

F. Jackson, in 1846, introduced crystal or sponge gold. In 1848 Thomas Evans made an amalgam composed of zinc and cadmium, which, however, was not a great success. The introduction of vulcanized rubber into prosthetic dentistry was due to Charles Goodyer in 1855. Dr. Barnum, in 1864, described the use of the rubber-dam for prolonged operations, an invention which produced great enthusiasm amongst his confrères, and a substantial recognition from them.

Then, in 1870, Morrison invented the dental engine with direct transmission of power.

In our own country we will only make special mention of Sir John Tomes. In the words of Dr. Geist-Jacobi, "he was the most renowned dentist of the nineteenth century, a man who belonged to the whole civilized world and not to any one particular nation." The memory of his life's devotion to the science and practice of dental surgery, and his untiring and successful endeavours to place our profession on a sound and scientific educational basis, is still so fresh in our minds that we need not dwell upon his work in detail.

In the earlier years of the nineteenth century there were a great number of dental practitioners in the British Isles between whom, however, there was no sort of professional communication or cohesion. It soon became apparent that, for the sake of the profession and the public, this state of affairs needed improvement. The first movement in this direction was made in 1841 by George Waite; from lack of support, however, his efforts proved fruitless, and for another decade no progress was made.

For the history of the reform movement in the dental profession I must refer you to the book on that subject Alfred Hill published in 1877. The strenuous and sustained efforts of an earnest band of reformers led to the formation of the Odontological Society in November, 1856, and shortly after its god-child, the Dental Hospital of London and School of Dental Surgery, was established in Soho Square. This was the first establishment of its kind to be founded in Europe. The efforts of this band to secure the recognition of the Royal College of Surgeons was crowned with success in 1859 by the decision of that College to grant a diploma in dental surgery.

Somewhat later by the fusion of the two rival bodies, the Odontological Society and the College of Dentists, into the Odontological Society of Great Britain, a happy termination was put to the acrimonious warfare over fundamental principles which unfortunately marred the infancy of the modern profession of dental surgery.

The steady and rapid progress which followed the foundation of our branch of the surgical profession upon a scientific, educational basis, culminated in the passing in 1878 of the Dentists Act, which has placed us under the protection and control of the law, and enabled us to expand on broad lines, and consolidate our ranks in professional security. This Act, although subsequent developments have proved it to have weak points, still offers wide potentialities for safeguarding our proper interests.

The greatest advance made in the nineteenth century was perhaps the discovery of the true nature of dental caries. The determination of its cause as a combined chemical and parasitic action was due chiefly to the remarks of Wedl, Leber and Rottenstein, Mills and Underwood, Black, and, above all, the brilliant work of Miller, of Berlin.

To conclude this sketch, I have extracted a few points of interest from the earlier British writers on dental subjects, hoping thereby to stimulate an interest in the many rare and curious books to be found in the Library.

In 1742 Joseph Hurlock, surgeon, published his "Treatise upon Dentition, or the Breeding of Teeth in Children." In it he describes the many ailments that infants are prone to during the dentition period, and enumerates many of the forms of treatment advocated by the ancients. The major part of the book, however, is devoted to an ardent advocacy of the practice of lancing the gums over erupting teeth. He reports in quaint language twenty cases, in which he claims to have greatly benefited infants by this treatment who were suffering from a variety of disorders, including smallpox, measles and scarlatina. One case is really amusing. He was called to a child, aged $1\frac{1}{2}$ years, suffering from convulsive twitchings, loose stools and retching; he promptly lanced the child's gums. Next day the child no better; lanced gums again. Next day heard the child was much better, so did not call for two days, when he was informed by the mother that on the night of the second day the child had passed large quantities of gooseberry skins and cherry stones—the nurse had confessed to allowing the child to eat about $\frac{1}{4}$ lb. of these fruits. He claims the improvement in the child's condition as due to his lancing, which shows him to have been somewhat lacking in sense of proportion and rather obsessed with his particular hobby.

"A Treatise on the Teeth," by A. Tolver, 1752. He describes how the teeth receive nerves and vessels at the orifices in their roots, and how these were frequently shown to the common people by the ignorant amongst the tooth-drawers as the worms which would soon have devoured the whole tooth had they not been skilfully removed. He emphasizes the necessity of sound teeth for health in masticating food and mixing it with the saliva. He also points out the dangers of hasty feeding and the use of liquid aliments to men of sedentary habits. All kinds of sweetmeats and sugars, he says, contribute very much to the destruction of the teeth, because their gluey juices stick to

the teeth; besides, sugars are of an acid, penetrant and corrosive nature. Those who love sugar and use it much rarely have good teeth—for instance, Dutch women. They are great tea drinkers; they do not sweeten it with loaf sugar as in England, but hold a piece of sugar-candy in the mouth which frequently serves for six to eight dishes of tea before it is dissolved, therefore it is very rare to find a Dutch woman of thirty or over with good teeth. He is very opposed to scaling, as it lays bare the necks of the teeth, and exhorts English ladies who have taken to this shocking practice to avoid becoming the dupes of foreign miscreants.

"A Treatise on the Teeth," Bartholomew Ruspini, surgeon-dentist, 1768. He says the causes of decay of the teeth are vapours arising from the stomach and lungs, keeping the head uncovered and exposed to the air and sleeping without a nightcap. He deduces that the particles exhaling from a carious tooth must be corrosive, as when the side of a tooth is decayed the contiguous one is affected, and when the top the opposite one which it touches in the act of closing is affected. Caries, he says, must be exposed and removed, and if the "cord" is opened it must be destroyed by the cautery or an instrument. "Such a cure cannot but be very torturing." When a "cord" has been destroyed the hole must be filled with lead or gold.

The first English book conceived and written in a scientific spirit is "A Treatise on the Disorders and Deformities of the Teeth and Gums," by Thomas Berdmore, member of the Surgeons' Company and Dentist-in-Ordinary to his Majesty, second edition published in 1770. In the preface he says: "When first I resolved to devote my whole time and attention to that part of surgery which concerns the dentist's art I observed with regret that no material instruction was to be derived from the writers of this country who have touched on this subject. Therefore I endeavoured for my own improvement to collect carefully from my predecessor in business and from practice whatever I thought conducive to the advancement of it." In this book is found the first mention in English literature of the use of the microscope in determining the structure of the teeth. He condemns the practice of transplantation of teeth, and says that if the replantation of teeth is so rarely successful, how much less can be the implantation of a tooth that can only roughly fit the socket and must press unevenly on the sides of the socket and inevitably cause pain and inflammation. He deprecates rapid extraction of teeth by mountebanks who advertise that they can take out a tooth quicker than other people can look at it; he likens the force used to that

of a blow from a hammer, and says it is a fruitful source of fracture of the alveolus. In an interesting chapter he describes some experiments he made to show the harmful action of many of the much advertised dentifrices of the period, and describes how rapidly deep friction grooves could be worn by a short period of daily transverse brushing with these powders and an ordinary toothbrush. We remember well the demonstration given to us 130 years later on the same subject by our late distinguished colleague, Professor Miller.

An interesting addendum to the book by Paul Jullion, surgeon-dentist, 1781, is his list of fees, showing as it does the conditions prevailing at that time:—

	£	s.	d.
Scaling the teeth	0	10	6
Bleeding and scarifying the gums	0	5	0
Filling or stopping up the cavity of a decayed tooth with lead	0	5	0
Ditto with gold	0	7	6
Filing and polishing a tooth	0	2	6
Extracting a tooth or stump	0	2	6
Transplanting a living tooth	5	5	0
Ditto a dead tooth	2	2	0
Engrafting the crown or body of a sound human tooth on the root of a decayed one	2	2	0
Constructing and fitting an artificial tooth with silken ligature	0	10	6
Ditto with gold wire fastenings	0	15	6
Ditto with gold spring	1	5	6
Constructing and fitting an upper or an under row of artificial teeth without fastenings	10	10	0
Ditto a complete set of artificial teeth with gold springs	25	0	0
Fitting and fixing a human tooth on the same construction as an artificial one with silken ligatures	2	2	0
Ditto with gold wire fastenings	2	7	0
Ditto with gold springs	3	3	0
Fitting an upper or an under row of human teeth on the same principle without fastenings	31	10	0
A complete set of human teeth with gold springs	73	10	0
Constructing and fitting an artificial palate	21	0	0
For the care and treatment required for preserving the health and vigour of the teeth, gums and contiguous parts of a person's mouth, at per year	4	4	0

The very much higher charges made for dentures containing human teeth conjure up before the imagination the horrible work of the body-snatchers and the high remuneration presumably extorted for their ghoulish trade.

In the preparation of this paper I desire to acknowledge the great assistance derived from two books—"Geschichte der Zahnheilkunde," by Geist-Jacobi, and "Notice sur l'histoire de l'Art dentaire," by Lemerle.

DISCUSSION.

The CHAIRMAN (Mr. C. F. Rilot) said the paper was one that did not lend itself to discussion, but should any member wish to speak upon it, it was open for him to do so. It was a most interesting paper, and there was not one line of it that they would have willingly missed.

Mr. F. J. BENNETT congratulated the Section upon possessing a Librarian who was so thorough in working up the resources of the Library. Those who had dipped into that inexhaustible treasure knew well how much pleasure and instruction was to be found in the noble Library that now existed. If he were inclined to be captious, he thought there were two names that should have appeared in the list of notable dentists. One important person in the history of dentistry was William Robertson, of Birmingham, who was the first to point out that caries was due to external action, and not to inflammation of the pulp. Anyone who would take the trouble to read Mr. Robertson's paper in the first volume of the Transactions of the old Odontological Society would find that Mr. Robertson not only dealt with the pathology of decay, but advocated dentistry in the Army, and referred to many other matters which were even now being discussed. Another name that should be mentioned was that of James Salter, who was also a pioneer. He thought the subject of the paper could be very much expanded by those who desired to follow in Mr. Densham's footsteps.

Mr. G. R. SHIACH said he had listened to the paper with very great pleasure, because in the previous week he had listened to a paper on the same subject read by Dr. Shennan at the Odonto-Chirurgical annual meeting in Edinburgh. It had struck him as a curious coincidence that he should hear two papers equally interesting but quite dissimilar within a week. Dr. Shennan, using lantern slides, referred to the early forms of instruments, so early that they seemed almost prehistoric. Both the lecturers noted that though early man was addicted to war, and had teeth very subject to traumatic conditions, extraction of the teeth was not a usual form of treatment, a rather surprising fact.

Mr. GEO. THOMSON said that although the paper did not lend itself to discussion, the members were very much gratified at the splendid paper Mr. Densham had prepared. He had been a little surprised to hear nothing of dentistry from the Chinese standpoint, because he had always understood that some form of dentistry was practised in China from the very early times. The paper had shown that there was a great deal to be gained by looking outside the profession of dentistry, because a great deal had been made known by the practitioners of surgery and medicine. He emphasized the value of the Library, for some of the superlative things said on our own speciality were to be found in such a book as Dr. Cavanagh's on the Care of the Body, in which appeared a very emphatic statement as to the importance of the care of the teeth, Dr. Cavanagh stating that a carious or decomposing tooth in the mouth was just as bad as a patient sucking a festering wound on the arm. Or, again, Dr. Savill, in the second volume of his work on Medicine, made the surprising statement in referring to the results of defective teeth that one owner of such teeth committed suicide owing to the condition of his mouth. There were many other books that were of interest to the dental profession, for example, Dr. Harry Campbell's book on Treatment, which showed very clearly that the teeth were of great importance.

Mr. W. HERN said one impression which the very interesting paper left upon him was the cosmopolitan character of dental surgery. From Germany came the suggestion for a plaster model, from France the first artificial substitutes for the natural teeth, and from another country wax impressions. America had added its quota to the list, but the paper disposed of any contention of one nation playing a chief part in the discoveries of dental surgery.

Mr. A. DENSHAM, in reply, thanked the members for the very kind manner in which they had received his maiden effort before the Section, and asked indulgence for touting for his own special branch, the Library. He felt that the intensely interesting nature of the Library was not fully appreciated by many of the members. The interest of taking up any line of investigation was that so many side-issues opened up, and with a large library to fall back upon there was every opportunity for pleasure and enjoyment, the difficulty being to know when to stop. It was a very difficult matter for a busy practitioner to read much beyond keeping abreast with the events of the passing day, but, as had been pointed out, it was a very desirable thing to realize that dentistry was not the narrow profession which some were content to regard it. It was good for a man's humility to realize how much he was indebted to other nations for benefits received. In preparing the paper he had been struck by the fact that almost all that was known of dentistry, except in minute details of technique, had come from the surgical and medical profession. Of all the earlier writers not one was a dentist pure and simple. He was very interested in the question of over-elaboration of dental conservative work that had

recently come before the attention of the profession, because he felt strongly the more that was known of general diseases the less likelihood there was of men devoting too much attention to the mechanical aspect of dentistry, and the more value would be given to the surgical aspect of that region treated by the dental surgeon regarding the oral cavity as it should be, as a part of the general system and not merely a happy hunting-ground of the constructive mechanician.

Odontological Section.

April 26, 1909.

Mr. LEONARD MATHESON, President of the Section, in the Chair.

Some Experiments on the Action of Formalin and other Root-dressings.

By HAROLD SIMMS, M.D.

IN recent years a great deal of careful thought has been expended on the question of the treatment of so-called dead teeth, and in consequence many new methods have been introduced and a large number of drugs extolled from time to time for their unusual efficacy in curing pulpless teeth that have become septic. With the possible exception of pyorrhœa alveolaris there is no domain in dental practice that is the cause of so much time spent—I almost said wasted—and in some fair proportion of cases fruitlessly spent, in combating the purely pathological condition of sepsis before we are able to restore, by fillings or by crowns, the direct results of dental caries. Therefore, as time is such a valuable factor in our profession it is little wonder that the improvements that have come about in our methods of treatment have been welcomed and made use of by those engaged in the practice of dentistry; and according as the results of the newer measures have proved superior to the older routine, so many methods are going rapidly out of use, and many of us are tending more and more to the adoption of methods similar to that introduced by Dr. Buckley, of Chicago.

For my purpose to-night it is not necessary to trace very fully the changes that have occurred, but you will all recall that we have had before us for many years drugs like peroxide of hydrogen, very valuable in oxidizing dead organic matter; drugs like pure carbolic acid and even perchloride of mercury 1 per cent., almost the prince of antiseptics;

drugs less powerful but more penetrating, like the various essential oils; and many other antiseptics, most of which, however, would fall into one of the above groups. We have had, indeed, a host of drugs to work with, and admittedly have had a certain amount of success with nearly all of them, but every careful observer will admit that he has come across many teeth that have not been amenable to any of these agents, and not only so, but that the cure of septic roots by such means has been a slower business considerably than it need be as a rule now, and that means that we have had to submit both our patients and ourselves to the inevitable loss of time that was formerly essential for the repeated dressing of roots.

From the fact that I have been discounting the utility of our ordinary antiseptics you will infer that I am advocating the use of formalin in some or other form as the most reliable, and certainly the most rapid means of overcoming sepsis following the putrefaction of the dental pulp.

Formalin is a 40 per cent. solution of formaldehyde gas in water, and is the basis of what is known in America as Buckley's treatment, and for those who are not familiar with his work I may say that his practice is to simply open up the pulp chamber of a tooth at the first visit, and to insert therein a pledget of cotton-wool soaked in equal parts of formalin and tricoresol, leaving the canals severely alone for the moment; he seals up the cavity, and after two or three days finds that the canals are free from sepsis, and he is at once able to proceed with the filling of the roots and the filling of the cavity itself if he so desires. In the vast majority of cases he found that a single treatment in this manner would cure any ordinary putrefactive pulp; the more complicated cases of fistula he found to demand, as we do, the syringing through of the fistulous track, but I am not at present dealing with this complication.

Although Buckley had found his remedy to be a very successful one, yet when he brought it before the profession there were many who were very sceptical as to the advisability of immediately sealing up a septic tooth, without even clearing out the root-canals. However, by cautious experiment it has been proved to be a thoroughly practical method, and the complaint of pain or the incidence of swelling subsequent to the tooth being sealed up is extremely rare. I have tried this line of treatment in a number of cases and personally found it very satisfactory, but my object here to-night is not so much to speak on behalf of Dr. Buckley's treatment as to bring before you a number of practical experiments on the action of various forms of dressings commonly

used by dentists. If my experiments do not show you any new fact, they will, I am confident, bring the problem of root-dressing before you in an entirely new light, and will, at least, help to explain how it is that the introduction of formalin has wrought such an improvement in the very practical subject of root-treatment.

My early clinical experience showed me not only the uncertainty and slowness of ordinary antiseptic treatment, but also the infinitely greater success that followed the use of the preparation doubtless familiar to you as * * *.¹ There can be no doubt that the manufacturers have early on fully appreciated the value of formalin, for to it is due the success, almost without exception, that attends the use of the host of proprietary preparations advertised so freely to the profession as abscess cures and the like. Having found out therefore that * * * in particular was a much more effective remedy than, say, oil of cinnamon, and this experience being confirmed by the opinions of other dentists, I set out to find on what principle this great superiority depended, and it is the result of this investigation that I have been carrying on during the last two years that I propose to bring before you very shortly.

I have mentioned the treatment of Buckley,² and it is convenient at this point to give you the explanation accepted by him as to why his tricresol and formalin is so effective. He claims that those two drugs succeed because they are able to convert chemically the contents of the septic root-canals into substances that are actually antiseptic. It is an ingenious and very simple explanation, and settles the question very comfortably. The precise chemical changes that are said to occur are the conversion of the putrefactive gases H_2S and ammonia into alcohol and urotropin respectively by the formalin. The fatty constituents of the septic material are changed by the tricresol into lysol, and thus all the main elements are converted into bodies that are antiseptic. These chemical changes have been worked out in the laboratory, and it is fitting that I should mention the existence of that theory, before passing on to consider how the experiments I am now going to describe may modify our acceptance of such a view.

The difficulty that confronted me at first was how to devise an experiment that would compare the antiseptic properties of ordinary fluids like carbolic acid, and at the same time those like formalin or * * * that claimed to accomplish their purpose by means of gaseous

¹ * * * For ethical reasons the name of a particular proprietary drug is omitted in this and following instances.

² *Therapist*, Lond., January, 1908, xviii.

exhalations. The late Dr. Miller made many experiments on the comparative efficiency of antiseptics, but all his work was done with reference to mouth-wash drugs, and he was able to give from the information he experimentally obtained an excellent formula for as nearly as possible sterilizing the oral cavity.¹ Now it seems to me that a drug sealed up in a root is not working under the same conditions as one in the mouth itself, therefore the methods used by Miller, which are the ones commonly adopted in testing antiseptics, do not seem capable of affording the information we want, for it is just as much the power of permeation and penetration as direct bactericidal strength that we want to compare when we are testing root-dressings. So I have not attempted so much to compare drugs according to their power of destroying microbes with which they are in immediate contact as to compare them in respect to their power of destroying microbes with which they are *not* in direct contact, and after a little thought I found out a way in which this can readily be done.

In describing the method I finally adopted for testing the various dressings, I must necessarily approach the matter a little from the bacteriological standpoint, yet the method is very simple and the technical points readily comprehensible. Essentially my method is to apply dressings to the roots of extracted teeth, seal them in, and drop the teeth into what are commonly known as plate cultures of bacteria; it is then a simple matter to watch the effect (if any) of the antiseptic dressings on the bacteria immediately around the apices of the teeth. As this is the whole key of my investigations, it is advisable that I should describe in rather more detail the exact proceedings in making such an experiment.

I first obtained a large number of teeth that had been extracted, single-rooted for the sake of simplicity, and cleaned out the canals thoroughly; with a little experience I found out that by excising the crowns of the teeth it was considerably easier to insert the dressings down the canals without placing any on the exterior of the teeth, and so complicating the issue, and, of course, the presence of the crowns of the teeth was in no way essential to the success of the experiment. I proceeded to take two of these teeth, and into one I placed a dressing of pure carbolic acid, and into the other a thin mix of * * *, both being sealed in securely.

The next step was to prepare a plate culture of bacteria, and for those who are not workers in bacteriology I may say that the usual

¹ "Micro-organisms of Mouth," Philad., 1890, p. 234.

method is as follows: A tube of agar culture medium is melted in boiling water, and into it is placed a platinum wire loopful of bacteria, and the tube is then well shaken up so as to get the bacteria diffused as evenly through the melted medium as possible. The tube is now rapidly emptied into a shallow glass dish specially made for the purpose and allowed to set, which it very quickly does. The particular microbes I used for this purpose were the common abscess staphylococci which I isolated from a septic root. In a plate culture the bacteria that are evenly distributed are unable to move once the plate has set, and when we place it in the warm incubator each little invisible microbe commences to multiply rapidly until within the space of a day a tiny speck known as a colony became visible to the naked eye, and is the expression of the growth of a single germ. Each of the original germs thus becomes a colony, and so the medium, instead of remaining nice and clear as when it set, becomes uniformly slightly or markedly opaque according to the number of organisms originally put in. To return to my experiment; into a plate culture prepared in the above way I dropped the two teeth containing, you will remember, dressings of * * * and carbolic acid respectively, and then placed the plate into the incubator, and after one day examined it to observe what had been the effect of the two dressings on the uniformly opaque growth, that I mentioned may be expected to be very readily visible in that space of time.

The result, you will observe, is really very striking, for while there appears to be a completely unretarded growth right up to the whole circumference of the tooth containing the carbolic acid, there appears around the apical region of the tooth with the * * * a clear area in which no growth has occurred, although this part of the surface of the plate contained its fair share of microbes when the plate was poured. It is evident now that in that circle, which is perfectly clear and transparent in the original preparation, the germs that were present have not been permitted to grow and thus become visible, but, on the contrary, have been destroyed, and that by a medicament sealed inside the root of a tooth; therefore something, obviously of a gaseous nature, must have passed through the apical foramen of that tooth; and if so that is surely a very striking fact, and one with which we have not up to now been familiar. It was only open to the possible suggestion that some of the * * * had been pushed through the apical foramen, and thus had the chance of direct action on the germs that I tried to eliminate. This experiment was therefore repeated in

precisely the same manner, except that particular care was taken that the * * * dressing was not placed more than half-way down the root canal, but the result came out exactly the same. Incidentally there may be observed in this preparation the excision of the crown of the teeth already mentioned.

Knowing that formalin is present in * * *, it seemed fairly clear from the above experiments that the advantage of that particular proprietary preparation depended on the presence in it of a drug that was not only powerful enough to destroy any germs it came into direct contact with, but was also able to sterilize the area immediately around the



FIG. 1.

* * * and carbolic acid. Black circle denotes area sterilized by the * * *.
Teeth are removed.

apical foramen, a quality that none but a gaseous antiseptic could possibly possess.

It would seem at first sight likely that the area of disease in a septic tooth is confined to the interior of the roots, but we know from the pathology of alveolar abscess that sometimes infection does pass through the apex, and I have, with further experience, come to the opinion that in those cases that either do not respond to the ordinary antiseptic treatment at all, or do so very slowly, there probably is a slight chronic infection of the peri-apical area, not of sufficient intensity to cause an alveolar abscess. If such be the case it will be perfectly

obvious that one might go on pumping down pure carbolic acid indefinitely without reaching the cause of the trouble, and hence the immense value of a formalin preparation which, from the above experiments, seems able to reach a point otherwise unapproachable.

The next experiment was on similar lines, except that while one tooth was again filled with * * *, the other was treated this time with the less powerful but more penetrating dressing oil of cinnamon, which is the most effective of all the essential oils. As may be expected, there is no difference in the result, and as before only the * * * gives a result showing positive penetration. The result of this experiment was also verified by its repetition. Amongst the essential oils I have tried are the oils of cloves, turpentine, eucalyptus, and also the combination known as Black's 1 2 3 mixture, which consists of the oils of gaultheria and cassia and carbolic acid, but none of these showed any evidence of their power

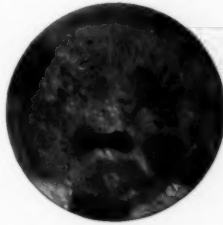


FIG. 2.

* * * and oil of cinnamon. Note lack of penetration by the essential oil.

to penetrate through the tooth apex. I next proceeded to try perchloride of mercury 1 per cent. solution, and the negative result of that and of Black's dressing may be seen in the next preparation. Neither iodoform nor the tincture of iodine gave any better result, nor did peroxide of hydrogen, although all the drugs so far named have been the subject of repeated trials. I have made a particularly careful series of trials with perhydrol, which is, as some of you are aware, a very strong preparation of peroxide of hydrogen, being just ten times stronger in the amount of oxygen it contains than the official 3 per cent. preparation. I was not, however, able to trace any antiseptic penetration through the apex. As a rule, my experiments were made after I had cleaned out the roots, but in the case of this drug perhydrol I made an additional trial in a root just extracted and full of septic material, so that there was abundant matter present to set free the oxygen from the perhydrol;

although, doubtless, the interior of the root was favourably affected by the oxygen, there was no beneficial result shown around the apex, for the organisms, as before, grew right up to the apical foramen itself.

Thus far my experiments seemed to show that no other dressing but * * * could act germicidally outside the limits of the canal in which it was placed, and the next step was to prove that not only * * * but its constituent formalin is equally capable of acting in the same manner. In the root that I am showing, which contains a dressing of pure formalin, you will be able easily to observe the large and perfectly clear area, in which every trace of bacteria has been destroyed by the formaldehyde gas given off from the formalin dressing. I am not suggesting or recommending that such a strong preparation as pure



FIG. 3.

Perchloride of mercury 1 per cent. and Black's 123 dressing. Penetration by neither.

formalin should be used in the mouth, for it is possible that irritant effects would follow; but for the purpose of demonstrating the great penetrative power of the formaldehyde gas I purposely employed an unusually strong solution. I employed a solution of half the strength in the next trial, and did so by mixing together equal parts of tricresol and formalin, thus coming to the formula that I have mentioned as originating from Dr. Buckley. This shows a little less penetration than pure formalin, but doubtless sufficient for the purposes we require, and when one looks at the result of this preparation it cannot but be suggestive of the belief that the excellent results obtained by means of this combination of drugs is due more to the power of wide penetration rather than to the complicated system of chemical changes that Dr. Buckley himself seems to have faith in. Tricresol is a body

very similar in origin and composition to carbolic acid, and, as may be expected, a separate trial of the penetration of this drug alone merely confirms still further the inefficiency of all liquid antiseptics for the purpose of treating dead teeth, and it becomes evident that it is only the formalin element in the combination of Dr. Buckley that is responsible for the advantages that follow its use. I attempted one trial with tricresol and formalin in a root in which I was not able to see with the naked eye any trace of an apical foramen, but it must have been there, for, as you can see, the gas found a way out.

From time to time I have tried various combinations of other drugs with formalin, both clinically and in this experimental way, and it does



FIG. 4.

No penetrative antiseptics by perhydrol.

not seem to matter very much what is the drug you dilute the formalin with, for I had quite successful results with each mixture I tried. One combination I tried was equal parts of formalin, creosote and glycerine, weaker still in formalin than Buckley's mixture, and * * * was used again as a basis to compare it with. In both cases there was a well-marked penetration, and it is convenient here to draw your attention to an occurrence that I was at first unable to account for—namely, the apparent escape of the formalin vapour not only at the apex but now and again at the other end of the root; however, after a time I noticed that this escape only took place in those roots in which I had sealed in the dressing with gutta-percha, so subsequently I sealed every dressing in with osteo-cement.

This double escape is still better shown in the next case, where I attempted to compare the penetrative powers of * * * and pure formalin; this preparation is shown as an illustration of the escape of vapour rather than as a real comparison of the relative efficiency of the two drugs named, and, as a matter of fact, I have not repeated that particular experiment.

From the foregoing work I think it may be conclusively asserted that no matter how strong liquid antiseptics are, they have no efficiency beyond the apex of the root they are placed in, but I am able to go a little further and show that both carbolic acid and oil of cinnamon will only kill the comparatively small number of germs with which they are



FIG. 5.

Marked antiseptic effect of pure formalin as shown by clear area.

in absolute direct contact. I am able to demonstrate the truth of that assertion by the following simple experiment. I made, as described, a plate culture of bacteria and let the plate set, and then placed on the surface of the medium three wisps of cotton-wool impregnated respectively with * * *, carbolic acid and oil of cinnamon, and then placed the plate in the hot incubator as usual. There is no gainsaying the fact that only in the area of medium surrounding the * * * wool is there the slightest trace of a penetrative antiseptic power, and that helps to establish still more strongly in my mind the view that these common liquid antiseptics are not able to perform effectively the work that we frequently require accomplished in our treatment of septic roots, and that as a rule therefore they should not be relied upon. My object

has been to indicate that we have at our command a drug of a gaseous nature that can be shown to have an energetic power of penetration and has also been proved clinically to be highly successful. There is no doubt that the class of case that we have found to clear up slowly under the old simple lines of treatment had no trace of infection of the apical area, and the gradual sterilization of the canals that was effected by, say, carbolic acid relieved the septic condition, but that does not alter the general fact that the vastly superior penetrative power of formalin is better able to sterilize root-canals that are often tortuous, and is capable both of curing the simple cases and also those of greater severity where there is a slight chronic disease in the peri-apical area.

The fact that we can obtain a very complete sterilization with formalin means that we are destroying the actual germs that are causing

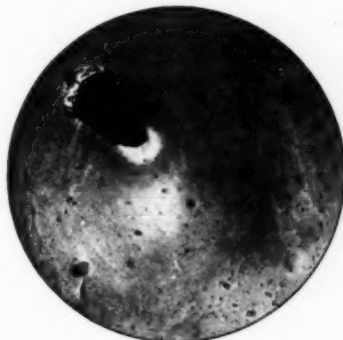


FIG. 6.

Equal parts formalin and tricresol. Note again clear sterile area.

the formation of putrefactive gases, and so setting up the pain common in septic roots; and it seems to be due to this thorough sterilization that we are able to seal up a septic root dressed with formalin on the first visit of the patient, instead of, as formerly, leaving in an open dressing. There may be some truth in the chemical theory of Buckley, but there seems to be also a strong case for believing that the generally admitted good results of the formalin mode of treatment are very largely due to the wide and thorough removal of the actual cause of infection.

I know that amongst a limited few there is a practice of using paraform as a root-dressing, and this substance is somewhat similar to

formalin in that it is equally able to give off the vapour of formaldehyde gas. Therefore when I tried the effect of a dressing of paraform in a root, it was only in accordance with what might be expected that a well-marked penetration through the apex was shown. Paraform, of course, is a powder, and so it is a very simple matter for those who fill roots with material like Fletcher's artificial dentine to incorporate a small proportion of paraform, say 5 per cent., in the permanent root-filling, and this will preserve the root in a very satisfactory manner.

It may be asked what preparation of formalin is to be particularly recommended for treating septic teeth, and it is an easy question to answer, for I have found almost every preparation I have tried clinically cures the sepsis and does so without setting up pain; I have tried two or three of the advertised abscess cures, and all of them were perfectly successful. I have also been successful with Buckley's tricresol and



FIG. 7.

* * * (above) and equal parts of formalin, creosote and glycerine. Both show penetration and the latter also shows leakage of dressing. Darker areas sterile.

formalin in equal portions, and equal success follows the substitution of creosote for the tricresol. I found, in fact, that as long as I kept to a solution of formalin, 1 in either 3 or 4, it was not of much consequence what I diluted the formalin with. It happens, however, that there are very few liquids that will mix with formalin, and so it follows that we are limited to either tricresol, creosote or glycerine. Therefore I can recommend for trial two parts of any of those drugs to one of formalin, and I am sure that you will find out that the clinical advantages of dressing septic teeth on these lines will prove to be as marked as they appear from the preparations I have been placing before you.

The permanent filling of roots after they have been freed from their sepsis is a matter of taste and difference of opinion, and I do not feel

that I can make any suggestion under this heading that you are not fully acquainted with; I may only just commend the suggestion I made in speaking of paraform that it is a very simple matter to incorporate in the root-filling some small proportion either of paraform or of formalin itself that will help to preserve the root in a wholesome condition.

Before I conclude I want to deal very briefly with another aspect of this subject of penetration—namely, the power of this vapour of formalin to penetrate along the track of the dentinal tubes. I have made one or two experiments that tend to show that just as cocain may be forced by direct pressure along the dentinal tubes, so it would appear probable that



FIG. 8.

Paraform. Clear area shows that formalin has been evolved and penetrated the apex.

when we apply paraform to a hypersensitive tooth the vapour of formalin penetrates the dentine a certain distance and in some way influences the sensibility of the irritable dentinal fibrils. When Mr. Spiller made a communication on paraform to this Society a little time ago he said that he considered that it made the teeth less sensitive because it probably penetrated a short distance into the dentine.¹ I have used paraform a good deal, and there is no doubt about the dentine being made less sensitive by it; in fact, the only question is whether the paraform does not do more than we require by penetrating to the pulp. Because I found a distinct use for paraform, but was a trifle uncertain of

¹ *Proc. Roy. Soc. Med.*, 1908, i (Odont. Sect., p. 41).

its ultimate effects on the pulp, I was rather glad to see a possibility of learning something about its penetrative power, and it was an easy matter to adapt the nature of my experiments to this purpose.

I took a tooth that was ground along its whole length, until I got about two-thirds of the way towards the pulp, perhaps a little more; I then filled the canal of that tooth as usual with pure formalin and sealed the tooth up. It was then dropped into another of the plate cultures of bacteria, already fully described, and again placed in the incubator. The plate after a day then presented a very interesting appearance, for there is plainly to be seen all round the centre of the tooth, and quite distinct from both ends of the canal, a distinct clear area containing no bacteria, and that circle is just in that part of the tooth that was most freely ground. From that it seems that there is a penetration of



FIG. 9.

Wisps of cotton-wool. Black area shows the spreading antiseptic effect of formalin, and the absence of it shows lack of antiseptic power of carbolic acid and oil of cinnamon.

formalin, at all events to a limited distance, in an extracted tooth. That was interesting, and showed that Mr. Spiller was probably correct in his view on the action of paraform, but we can get a little nearer the truth still. I selected two teeth, filled the canal of each again with formalin, and then thoroughly sealed up both ends of both teeth with cement. These teeth were treated exactly as before, and the plate culture in which they were dropped was incubated for a day. There was this important difference between the two teeth; one was not ground at all, while the other was ground half-way to the pulp, but only in the middle third of the tooth. The idea of this step was to determine first whether the vapour of the formalin would penetrate the half thickness of dentine left in the ground tooth, and in the second place

whether there would be a penetration through the full thickness of dentine plus the cement. However, it is quite clear that there was penetration only in the case of the ground tooth, and we are thus able to assert that in an extracted tooth strong formalin will penetrate at least half-way through the dentine; I wondered whether the experiment made with the unground tooth was going to help the theory that there is a way of communication from the pulp through the dentine and cement to the outside, but my result being quite negative, as you may see, gives no support to that theory, and even if there be a communication it is evidently not of much consequence if it will not permit the passage of a fine vapour.

Referring to the practical use of paraform, I have made two further experiments which afford some information. I placed in a root-canal

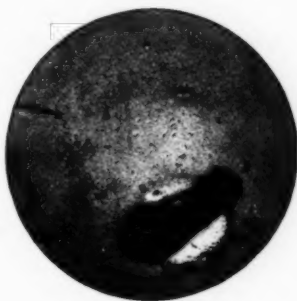


FIG. 10.

Tooth ground in middle third and containing formalin. Shows that the vapour can penetrate dentine along track of fibrils. Both ends sealed up.

paraform on cotton-wool of the strength in which it may be used in order to treat sensitive teeth—namely, about 1 in 10 of zinc oxide, adding a drop of eugenol merely to make a paste. This particular tooth in its middle third had the coating of cementum and a trace of dentine beneath the cementum ground away. It was completely sealed up at both ends and then incubated in a plate culture as before; in this strength and through this thickness of dentine there appears to be no penetration of the formalin vapour from the paraform, and a very fortunate thing it is for those of us who are in the habit of using paraform. We have arrived at the position that weak paraform cannot penetrate a very thick layer of dentine, even in an extracted tooth, and I imagine it would be still more difficult to penetrate far in a living

tooth where there would surely be more obstruction. It merely remains now to show that this weak paraform can penetrate a thin layer of dentine. By repeating the last experiment, with the exception of grinding about two-thirds of the way to the pulp instead of only a trifle, it was easily demonstrated that this small amount of penetration really can take place. These experiments give us some information about the action of paraform, and also give us a distinct warning not to use these formalin preparations in strong solutions or when the cavity we are preparing approaches anywhere near the pulp. I think, however, we are justified in some of the more troublesome cavities—shallow cervical ones, for instance—in making use of paraform, which is a very real help to our patients in such cases.



FIG. 11.

Top tooth unground and lower one ground; formalin in both. Shows that formalin can penetrate dentine, but not dentine plus cementum. Both ends sealed up.

In thanking you for listening so patiently to this communication, dealing with the scientific aspect of some of our everyday problems, I would like in conclusion to say that I am not so much asking anyone to adopt my personal lines of treating septic and tender teeth, but rather of placing before you impartially the results of the experiments I have been making, with the idea of elucidating the problems that have arisen since formalin came into use, as to how and why it gave such good results and exerted such a powerful influence on teeth, both septic and sensitive. If you follow the literature on these subjects and at the same time watch, as I have recently done, the enormous number of new proprietary preparations being constantly brought before

you for the treatment of these dead or septic teeth, and all of them expensive and relying on nothing more or less than formalin to do their work, if you watch these points you must agree that the examination carefully of the action of formalin is a most important matter, and I am very pleased to have had this opportunity of bringing before you some facts connected with this powerful drug. It is always a very satisfactory thing when we find a new drug or a better method of treating disease, but it becomes very much more satisfactory when we learn something of the mode of action of such a drug or method, and why it should be better than the methods that have gone before it. That is my excuse for this paper, and I am indebted to your research committee for the means of carrying on the series of experiments, which have to me been a most interesting study.

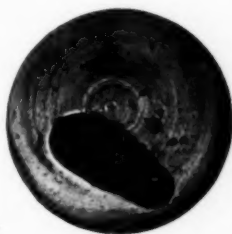


FIG. 12.

Tooth cementum ground away in middle third; paraform, 1 in 10 of zinc oxide in canal. Shows no penetration through full thickness of dentine.

DISCUSSION.

Mr. STANLEY MUMMERY had had the privilege of reading the paper and was specially interested in it, as he had used formalin for about eight and a half years in the treatment of dead teeth to the exclusion of all other antiseptics. The experiments afforded a scientific proof of methods that hitherto had been entirely empirical. No other antiseptic was known that would penetrate dentine in such a way, and the use of paraform had shown that a considerable degree of penetration did take place. He had been rather surprised at the supposed danger of using formalin in full strength, because he had never used it in any other way for the last eight years, and had had no trouble at all, the worst that had occurred being very slight pain immediately on the application of the drug, and that had passed off at once. His practice had been to swab out the septic canal with formalin and then insert a wisp of wool soaked in

formalin for twenty-four hours. He would be glad if Mr. Simms could say anything about the composition of paraform. He thought there was a large field for further investigation of formalin in the treatment of inflammatory conditions of live pulps. In weak solutions formalin acted not as an irritant, but as a soothing agent, and he thought it might be very useful in cases of partially exposed pulps or very nearly exposed pulps in which some bacterial invasion had taken place.

Mr. H. BALDWIN said since Mr. Spiller introduced formalin to his notice he had been in the habit of using it as an obtundent, about 5 per cent. mixed with Fletcher's dentine or with dressings, and he had never had any reason to regret it, except occasionally when using it a little too strong there had been considerable pain as a result. He had never known any lasting trouble set up, and had never known it to kill a pulp or be followed by periodontitis. As a root-filling he used chloropercha and paraform with a little cotton-wool dipped in it to enable it to be pushed down the canal. The results had been very good and no irritation or trouble had ever followed. The paper of Mr. Simms he thought a very useful one, especially as demonstrating the penetrating character of the formic aldehyde gas as evolved from formalin or paraform.

Mr. F. J. BENNETT thought the author had made a slight mistake in saying that Dr. Miller had not performed any experiments on teeth in a similar manner. He believed the first public appearance of Dr. Miller in England was at the National Dental Hospital, when he read a paper on the subject of antiseptics applied to root canals. That would not appear in the *Transactions*, but was no doubt to be found in some of the dental journals.

Mr. J. G. TURNER said the particular point that interested him was the penetrating power of formalin. He had used it just as it was bought for a long time in treating septic roots, and had been always expecting that some day it would penetrate the apex and he would be sorry for using it. He had never discovered, however, that there was any irritation. He had been wondering why formalin did not penetrate to the apex, because sometimes it must have been put more closely to the apex than was thought, although as a general rule one was further away from the apex than one thought! He was also wondering whether formalin penetrated so readily amongst putrid germs and their products as along dentinal or root canals in which the protoplasm was either dead or cleared away, or only just separated from its vital attachment, and so readily mummified and shrunk by the action of the vapour, in which latter case the penetration was not comparable with what was desired. What was desired was penetration along septic root canals filled with putrid matter, and along dentine canals filled with germs. He thought Mr. Simms had said that he cleared out the roots in his experiments, and in that case he was afraid he provided no answer to the question.

Mr. PEYTON BALY said about a year ago he had a case in which he treated a distal cavity of a second upper molar with about $\frac{3}{4}$ gr. of paraform applied on a small wisp of wool with Fletcher's carbolized resin. The pulp was septic and he had no time to do more than get a burr into it. He saw the patient a

week afterwards and there was a good deal of pain. On excavating the tooth he found carious dentine well below the gum level and a large amount of necrosis of the alveolar bone between the second and third molars, and eventually he had to take out the third molar in order to get the necrosed bone away.

Mr. GEORGE THOMSON, in support of what Mr. Baly had just said, showed a tooth he had to extract together with a ring of necrosed bone surrounding it. The canals had been treated with paraform three or four weeks previously.

Mr. SCHELLING asked Mr. Simms whether he had found nascent oxygen absolutely useless, and, if so, whether peroxide of hydrogen had not the properties attributed to it.

Mr. HAROLD SIMMS, in reply, said most of the remarks referred to the use of paraform in the treatment of sensitive teeth, and that was only an after-thought in the particular work he had been doing. With regard to the use of formalin in full strength, he had never had the courage to try it. He had always understood it was a dangerous thing, and as he obtained equally good results from weaker solutions he had never tried full strength. He had no doubt it was quite satisfactory, and would try it when he had an opportunity. Paraform was stated to be simply a polymer of formalin, a multiplication of the same chemical atoms in the same proportions but with a larger number of them bound together. He had been interested to hear that formalin might be used as a dressing in teeth with live pulps, because that was a point Americans who had written on the subject had been dead against, as they believed it would set up an immense amount of irritation, partly on contact, and later on. He had not tried it in live teeth at all. He did not want to minimize the work of Dr. Miller, because he knew Dr. Miller had done more than anybody else on scientific lines for the advancement of dentistry, but he had been through Miller's work and he thought the fact he had stated in the paper was true, that most of Miller's work referred to drugs used for the purpose of preventing decay. Dr. Miller might have done a certain amount of experimental work on root treatment, but the methods he adopted were the same that he adopted for testing mouth-washes. Such methods were of little value in this connexion, because they did not take into account the permeation of antiseptics, but merely the effect of any particular antiseptic mixed up with a certain proportion of germs with which it was in direct contact. He had no doubt there was less penetration in teeth in the mouth than in the particular preparations he had shown, and probably that was a good thing, as it was not necessarily a very advantageous thing to have formalin penetrating widely through the apex into tissues partly diseased but with tissues near them quite healthy, as in that case it might do more harm than good. No doubt there was some penetration even in the mouth, but much less than in the preparations. In fact, he was quite sure there was some penetration in some cases, because now and again cases of chronic abscess and of fistula were occasionally cured simply by placing formalin in the roots, without syringing through at all. No doubt in cases of chronic abscesses and fistula there was more of a definite opening through

the apical foramen than there was normally. He was himself surprised that the action of nascent oxygen was not better marked, especially in the case of perhydrol where there was undoubtedly a large quantity of oxygen given off at the time. He repeated the experiments a number of times, but even if oxygen came through the foramen it did not have any effect at all, because the germs grew right up to the teeth, and there was no antiseptic action of the oxygen. He was not so sure that oxygen would absolutely prevent the growth of the germs in a culture medium; a great many germs only grew in an atmosphere of oxygen. On the other hand, there was the idea that oxygen was an antiseptic, although its advantages were probably more in the way of getting rid of organic products of bacteria rather than destroying the actual bacteria themselves.

A Case of Round-celled Sarcoma of the Lower Jaw.

By HAROLD CHAPMAN and E. ROCK CARLING, F.R.C.S.

L. D., AGED $3\frac{1}{2}$; round-celled sarcoma of right lower jaw; excision of half inferior maxilla, March 24, 1908. *Vide* Case Book—"Surgical Cases, females; 1908"—of Mildmay Mission Hospital, Bethnal Green.

Mr. CARLING said he wished to ascertain if anything could be done to improve the appearance and powers of mastication of the child. About fifteen months ago she came to the out-patient department of the Seamen's Hospital because the mother was not satisfied with the doctor's diagnosis of a swelling at the right angle of the jaw. The case was seen by several people, by Sir William Bennett and Mr. Carless, neither of whom would express a definite opinion, although the general opinion seemed to be in favour of tuberculosis of the glands and not in the jaw itself. In the course of about three weeks it became perfectly evident that the jaw itself was the seat of the tumour; there was very considerable pain, generally over the whole fifth nerve distribution. About a month later, as egg-shell crackling had become evident, he excised very nearly half the jaw, beginning in the neck, taking up the lymphatic glands and the salivary glands in one mass with the bone and going up to the condyle. On microscopic examination it was found to be a round-celled sarcoma, and on that diagnosis he anticipated the child would die within the year, but it was now fourteen or fifteen months since the operation. As the child appeared now to be in perfect health it seemed very important that something should be done, and it was with the view of getting opinions as to whether anything could be done that he asked Mr. Chapman to show the case that evening.

Mr. CHAPMAN said the patient was brought to Guy's Hospital about October last, six months after the operation, but she was then almost a baby

in arms, although 4½ years old, and it was quite impossible to do anything. She was seen again about a month ago and impressions were taken. It was found that the lower jaw moved very considerably towards the side from which the portion had been removed. The patient was now 5 years old, and her mother said she could eat perfectly well, though the occlusion showed mastication to be impossible, so that perhaps it seemed a little doubtful whether any treatment should be adopted at all. The jaw could be pushed over without any difficulty until almost normal occlusion could be obtained. It might be expected that a good facial result could be obtained by using some appliance in the upper jaw bearing on to the lower teeth and holding them in occlusion with the upper ones, but to accomplish that it would be necessary to have a head bandage so that the mouth was kept continually closed or for a long period in each twenty-four hours.

Mr. J. LEWIN PAYNE thought in such cases much might be done to prevent deformity by the introduction of some interdental splint in the early stages—either immediately after the operation or within a few weeks. By that means the relative position of the mandible to the maxilla could be maintained and the comfort of the patient was increased. By employing a skeleton splint it was quite possible for the surgeon or the dresser to cleanse the parts sufficiently without any additional risk of sepsis, and apart from this it seemed to him that the use of an interdental splint favoured union after operation, in that the mandible was kept in a fixed position. In the present case the suggestion Mr. Chapman had made as to using some form of appliance in the maxilla with an inclined plane seemed the one most suitable to adopt. The jaw was quite movable at the present time, and the remaining teeth could be brought into accurate occlusion, and if means were adopted now to keep the teeth in their relative position the result should be quite satisfactory.

Mr. JAMES thought a case he had shown in the Section about a year ago bore somewhat on the present case. There was extensive necrosis on the right side of the mandible, causing the removal of a large part of the bone, including the condyle; the loss was almost as extensive as in the case now under discussion. He made several efforts to do some of the things that had been suggested, but was defeated, partly because the boy had a difficulty in attending the hospital. Since then the teeth had erupted in such a way that they articulated even although the mandible was pulled across considerably to the right side, and he felt now it was as well to leave it alone, seeing that a certain amount of useful mastication was available. He thought if he tried to put in a splint it would be exceedingly difficult, as the work would extend over a long period and it would mean making a large number of splints while the mouth was growing. He was not sure that what had resulted in his case unintentionally was not the best thing that could have happened.

Mr. J. F. COLYER thought in such a case the wisest course was to leave the child alone, especially as it was thriving fairly well, because the introduction of any appliance into the mouth would necessarily assist any sepsis that might take place. Only that day he had seen at the hospital a man who had

half the mandible removed in a bad case of epithelial odontoma, and there was very little displacement of the remaining half of the mandible. With the slightest amount of pressure the two teeth the man happened to possess in the mandible could be brought into correct occlusion. Some time ago a plate had been put in with a spring on the affected side to push the mandible across and correct the occlusion, but within two months he had lost the two teeth, and that seemed as though it must always be the case in trying to restore a fragment; it was acting against cicatricial tissue and sooner or later the teeth gave way. He removed the two teeth and put in another plate, taking all the bite off the side where the jaw had been removed, but even then the patient came back with a certain amount of ulceration on the free margin of the mandible, showing quite clearly, even with all the care exercised, the constant tendency of the portion of the mandible to come round to the affected side and become rubbed by the denture. A case of the greatest interest was that in which Mr. Boyd removed half the mandible of a boy aged about 14 for a myeloid sarcoma, and Mr. Colyer started by putting plates to counteract the tendency of the remaining fragment of the mandible to go over. Within about three weeks the boy had two of the teeth in the remaining portions of the mandible loose, and the splint was taken out and the mouth left alone. When last heard of, about six months ago, there had been no recurrence and the boy was well nourished. If the splint had remained it might have set up sufficient irritation to lead to a recurrence of the disease.

Mr. COLEMAN referred to a case of cystic tumour of the mandible, probably an epithelial odontoma, which he had seen at St. Mary's Hospital a few weeks previously, and in which a portion of the mandible was excised from the first premolar to the angle (jaw). Mr. Dolamore inserted a splint (fixation), and the case looked very promising, although it was too early to say what the final result would be. So far there was not the hideous deformity generally seen in such cases after operation. Later on Mr. Dolamore hoped to put in some permanent apparatus carrying the missing teeth. It would appear as though the time of the operation, as in the above case, was the right time to apply any apparatus to prevent resulting deformity.

Mr. CHAPMAN said the question of having a splint made before the operation was not overlooked, but the latter had to be performed rapidly once the diagnosis had been made, the question of time being a very important point. For this reason nothing was done. His own idea was an appliance to endeavour to hold the jaw somewhat in its correct position, but after having heard the discussion that evening he felt a little doubtful whether that was the best thing to do. Mr. Payne had suggested to him that he was in favour of using a crib apparatus fitted in the upper jaw with an inclined plane, against which the lower teeth would bear and so bring the lower jaw into position. The difficulty in the case was the age of the patient, which rendered it almost impossible to adjust satisfactorily any apparatus in the mouth; moreover, the teeth were deciduous and therefore would not stand much pressure.

Odontological Section.

MAY 24, 1909.

MR. LEONARD MATHESON, President of the Section, in the Chair.

A Preliminary Note on the Eruption of the Teeth.

By W. WARWICK JAMES, F.R.C.S., L.D.S.

I WISH to bring before the Section to-night a few facts which are, I think, of importance with regard to the eruption of teeth. Theories have been formulated to explain this phenomenon, but, as they do not bear directly on what I have to say, I shall not discuss them. My investigations have been made of the tissues over the teeth previous to eruption; the parts examined have been in connexion with the teeth of the temporary dentition, chiefly the incisors and canines. It has long been known that the soft tissues associated with the teeth contain certain epithelial remains of the toothband and enamel organ which do not exercise any known function. I am about to show that these remains are not lost at so early a date as is generally thought, but are present up to the period of eruption and are the determining factor in directing the tooth to its final position in the jaw. I propose in this paper to give first a résumé of what is known with regard to these elements as far as they concern us. I shall then deal with the subject, which applies entirely to the tissues after birth, under the following headings:—

- (1) The character, arrangement, and sources of the epithelium.
- (2) Changes occurring in the epithelium and in the connective tissues.
- (3) Importance of the epithelium in the eruption of the teeth.

RÉSUMÉ OF FORMER ACCOUNTS CONCERNING THIS EPITHELIUM.

Previous investigations into the histology of these remains have been almost entirely restricted to foetal tissues. After birth they have been considered chiefly from the standpoint of pathology. In prenatal periods they have been thoroughly investigated and described by Malassez [1] [2], who also quotes freely from the former work of Legros and Magitot. He finds numerous buds which are derived from the gingival epithelium (gingival buds), from the epithelial lamina, and from the external surface of the enamel organ. These buds, with parts of the dental lamina, epithelial cords, and external epithelium of the enamel organ, exist as epithelial remains, forming three principal groups:—

(a) Superficial group adjoining the deep face of the gingival epithelium.

(b) An intermediate group or intermediary plexus.

(c) A deep group in connexion with the external surface of the enamel organ.

The superficial group is composed of the gingival buds and prolongations of the epithelial papillary processes. The cells are usually of the Malpighian type, the peripheral ones often being cylindrical. Sometimes the central cells are extended and flattened as "*globes épidermiques*." They are probably certain epithelial products of the Malpighian layer which Serres has described as dental glands.

Of the intermediary plexus, epithelial products are found in the thickness of the gum between the mucosa and the dental follicle. They appear to extend along the whole of the gingival ridge, and in transverse sections may appear as large as the follicles. They are very irregular in thickness and density; the masses vary in number and in continuity; in places they are nearer to the mucosa, in others nearer to the follicles. A certain number appear on section to be independent of each other—perhaps some are so in reality—but the greater part anastomose, so that one may consider them as forming a very irregular epithelial network.

The cells are mostly polyhedral, but of no definite type, sometimes spherical or irregular. Occasionally they are cylindrical when they form an outer layer, the inner ones being polyhedral or spherical, and may be so arranged that the columns appear to be tubular. This intermediate network communicates here and there by cords of polyhedral cells with the gingival epithelium and also with the enamel organ. This epithelial plexus is probably the major part of the epithe-

lial lamina (toothband) which has budded, and which has been broken up and displaced by the growth of the surrounding tissues. The buds from the enamel organ also probably play a part in its formation.

The third and deep group of epithelial remnants is derived from the external epithelium of the enamel organ and the buds proceeding from it. The buds were probably recognized by Hérissant [3], and are perhaps what he thought were glands which secreted the enamel. Todd and Bowman described them and compared them to glandular formations. Robin and Magitot [4] gave a much fuller account of them, and later they were again dealt with by Legros and Magitot [5]. The external epithelium of the enamel organ does not form a continuous layer, as is generally believed, for on tangential sections in certain places numerous gaps are to be seen very clearly, which give the appearance of an epithelial network. This network is interlaced with vessels, so that where the windows exist in its meshes, the latter come into contact with the enamel pulp (stellate reticulum). The cells are flattened, their long axes corresponding to the long axes of the processes. At points where no network exists the external epithelium is a continuous covering and is generally thicker, and also composed of more or less flat cells. The buds which start from the external epithelium, either where it exists as a network or as a continuous layer, are formed of polyhedral cells; the deep cells are flat, like those of the external epithelium, but at the extremity of the bud they are more cubical. They resemble the intermediary network, but globes épidermiques are not seen. From this account cylindrical cells are present to a greater extent than I have been able to find them in my sections which are from postnatal subjects, whilst Malassez's sections are from fetuses between two and a half to six months. Legros and Magitot state that "the composition of the band is quite simple at first: it is composed of a central layer of small polygonal cells surrounded by a continuous row of prismatic cells;" but later, in reference to the epithelial remains, they say: "Never do we find in the debris of the cord or band the prismatic elements." It is of interest that in my sections the lower part of the toothband above the permanent tooth shows cells of a cylindrical character.

Legros and Magitot describe the dental follicle as becoming isolated in the jaw, the epithelial cord being ruptured, so that it is no longer directly connected with the mucosa. This rupture of the cord is placed in their table as occurring at the fourth month. Further, they say: "The epithelial lamina, once deprived of its continuity with the

follicle, becomes the seat of a marked proliferation of the elements which compose it." And, later: "Whilst the formation of buds is going on at the expense of the remains of the cord, similar changes are occurring on the external surface of the enamel organ. It is at the highest point of the enamel organ that these buds are most abundant, diminishing in number little by little upon the sides. They anastomose laterally and also with the buds of the cord, so that an irregular network is seen." They therefore seem to imply that the primary connexion is lost, and a secondary one established by the anastomosis of the buds.

From the above accounts it would seem that the formation of the toothband and its processes which give rise to the enamel organs is far less simple than one is led to think. The usual descriptions lead one to believe that with the formation of the enamel organ the toothband has performed its function and undergoes atrophy. Yet I venture to suggest that the proliferation and the formation of buds indicate a continuous growth which continues with variable activity until eruption of the teeth takes place. It is true the above descriptions apply to the foetus and it is also true that much of the epithelial tissue undergoes atrophy. It is to the proliferation and atrophy of this epithelium that I wish particularly to draw your attention. I will first refer to Röse's paper [6].

Legros and Magitot published their researches in 1873. In 1885 Malassez's paper appeared, and in 1891 Röse, using Born's method of modelling, showed more accurately what occurs. With regard to the toothband he says: "The forepart becomes modified, showing marked thickenings, whilst the intervening parts are attenuated and in places separated in their continuity. This process goes on continuously from before backwards, so that at birth and later the sections show that the front part of the band consists only of apparently disconnected epithelial fragments, whilst the back part is still a smooth and an unbroken band" (shown well in his models). He describes the external epithelium as showing marked proliferation near the apex of the tooth, where it forms low papillæ-like outgrowths. These increase in size and number; between them interruptions occur in the continuity of the epithelial cell layer. With the changes which cause the toothband in front to become sieve-like, this structure's connexions with the surface epithelium have been partly severed; similar changes occur in the connecting bridges, but in a much higher degree.¹ On section these are generally

¹ Röse objects to the term "neck of the enamel organ," as there is frequently more than one cell column connecting the enamel organ to the toothband, and suggests the term "connecting bridges." There may be only one for those anteriorly situated.

seen, as is often the case with the toothband, existing as apparently disconnected fragments of epithelium. The connexion of these as a continuous network was first shown definitely to be so by the reconstruction method. Later, epithelial fragments become separated and lie free as the tooth-band becomes more broken up. They may persist unaltered, or enlarge to form epithelial pearls or little cysts.

So far, with a few exceptions, the accounts given refer to the conditions found before birth. With regard to the fate of these epithelial products, Legros and Magitot state: ". . . all end by being entirely absorbed and disappearing." It is probable that such statements as this have led to the view at present generally accepted, which is summarized by the following statement by Mr. Hopewell-Smith [7]: ". . . nothing intervenes between the oral epithelium and the stratum intermedium but a large amount of submucous tissue composed of long branching fusiform connective tissue-cells imbedded in a thin stroma, which also contains blood-vessels and at times tiny masses of epithelium (glands of Serres). The latter are derived from the fenestrations of the tooth-band."

The two following statements concerning the epithelial remains over the permanent tooth are of importance. Röse says: "This leading string (*gubernaculum dentis*) consists entirely of connective-tissue fibres which are separated by epithelial remains of the toothband. It has no physiological significance." Malassez also says: "In the *gubernaculum dentis* there is no canal, properly speaking; but there are numerous epithelial cells. Their direction is longitudinal, like the connective tissue. In the lower parts they are more abundant and more voluminous; they anastomose frequently between themselves, and form thus a rich network; besides this, they show in places kinds of lateral buds in the form of bundles."

The following from Legros and Magitot is of particular interest, for they recognize the atrophic changes which occur at the period of eruption: "In the human embryo the remains of the cord of the temporary follicle are found almost beyond the time of the formation of the permanent follicles, and it is probably during the movement of eruption that the buds wither. It is nearly the same in the dog. In the calf and the sheep it seemed to us that the disappearance was earlier; but we believe that generally it is towards the time of eruption that it is complete. The physiological meaning of these phenomena appears difficult to determine."

I now proceed to deal with the subject under the headings already enumerated:—

THE CHARACTER, ARRANGEMENT AND SOURCES OF THE EPITHELIUM.

I wish to establish the following points:—

(a) That the epithelium is produced continuously up to the period of eruption.

(b) That there is probably a continuity of epithelial tissue extending between the surface epithelium and the remains of the enamel organ.

(c) That the epithelium is derived from three sources: (1) The external epithelium of the enamel organ. (2) The toothband and the connecting bridges. (3) The surface epithelium.

(a) In order to establish this first point, my sections from infants of two weeks and onwards show that previous to eruption the tissues overlying the tooth contain epithelium in considerable quantities and that it is distributed in a similar manner to the epithelial remains in the foetus. There is much variation in the character of the cells according to the stage of eruption at which the tooth has arrived, but the general distribution is the same. As we have already seen, with the growth of the tissues the toothband has become broken up so that the arrangement becomes more in the form of cords or columns; some of the cells undergo atrophy, but associated with this is the formation of buds and an increase of the epithelium. The latter must have occurred if my second point is correct, for with the growth of the neighbouring tissues the enamel organ and the surface epithelium have become further separated, and in order to maintain the connexion by epithelium between these proliferation must have occurred. There is also a cap of epithelial cells over the erupting tooth, to be found at all stages, which must also have proliferated in order to accommodate the increase in size of the crown of the tooth. In the earlier specimens very large collections of epithelium are found passing in from the surface epithelium and are possibly directly derived from the toothband as they form a distinct line running parallel with the gum ridge.

(b) The second point can only be definitely established by the reconstruction method (Born's models), as has been done by Röse in the earlier periods. I have endeavoured to determine it by cutting series of transverse sections, commencing from the gum ridge and examining each section until the tooth is reached. In all these sections collections of epithelial cells are found, but varying in size and number. Those sections nearest to the surface show the epithelial interpapillary processes cut across. Immediately below this the epithelium is very variable in

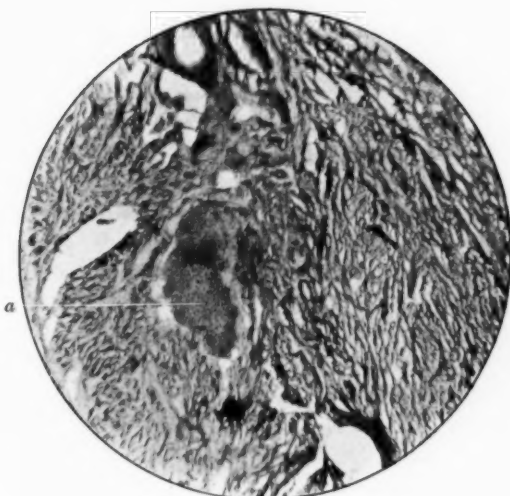


FIG. 1.

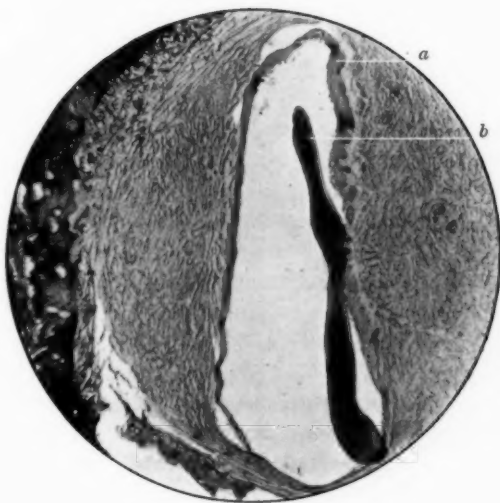


FIG. 2.

Figs. 1 and 2.—Two transverse sections from a series which showed the epithelium to be more or less continuous between the surface and the tooth. (1) epithelial column, *a*, cut across; (2) epithelium lining sac, *a*; tooth cut across, *b*.

amount and its continuity is sometimes difficult to trace, the collection of cells frequently being very small, but when this occurs they are usually numerous. There may be one column which is quite definite. In one of my series this column is connected with the surface epithelium a short distance below the gum ridge and stains somewhat differently from it. In following the epithelial columns from section to section, besides the variation in number one does not always find it exactly in the spot one seeks for it. This coincides with what one expects from vertical sections, for here the columns are nearly always cut across, appearing as a rounded group of cells, and only occasionally does one find them so as to appear as a cord cut longitudinally. The tortuous nature of the column accounts for this. The permanent toothband, which has undergone less change when seen at the same time in vertical sections, is of this character. In some of my sections it can be traced more or less throughout its whole length on the same slide. The column of cells can be followed until the epithelial cap which is surrounding the tooth is reached. In one of my series the column passes directly to the top of the epithelial cap, but in others it passes to the side of it and appears to be connected by numerous small columns. This variation is probably due to the difference in the state of eruption at which the tooth has arrived. In the former the tooth was very near the surface; in the latter eruption had hardly commenced. In Röse's models the toothband is not connected near the apex, but slightly to one side. In the neighbourhood of the tooth the tissues contain more epithelium, chiefly as small collections of cells. The cells are mainly polyhedral in type or somewhat flattened, until the epithelial cap is reached, which I shall describe shortly, as well as the changes which can be seen in the cells themselves. Columns of cells can be traced further, for they pass to one side of the temporary tooth to become eventually connected with the permanent tooth germ, as is well seen in vertical sections. In two of my series of transverse sections I was able to cut vertical sections of the corresponding tooth of the opposite side and so compare them. The sections which involve the tooth show a definite layer of epithelium lining the inner side of the follicle. It has been recognized for some time that the cap of tissue covering the unerupted tooth is lined by epithelium, but no particular importance has been attached to it. Dr. Paul [8] recognized its nature, and Mr. J. G. Turner has recently drawn attention to it. It is difficult to obtain sections without displacing this layer, but it is possible to determine its nature from the parts which are here and there intact. I do not mean it is impossible to get the complete ring of epithelium

surrounding the tooth, but its relationship to the neighbouring tissues is often disturbed and the innermost cells are frequently removed.

(c) The epithelium is derived from three sources, as enumerated above, and corresponds closely to the description already given as occurring in the foetus :—

(1) *The External Epithelium of the Enamel Organ.*—On examining sections at different stages the epithelial structures lying immediately over the tooth are found to vary with the period of development and the degree of eruption. When the enamel organ consists of an internal epithelium, a stellate reticulum, and an external epithelium,

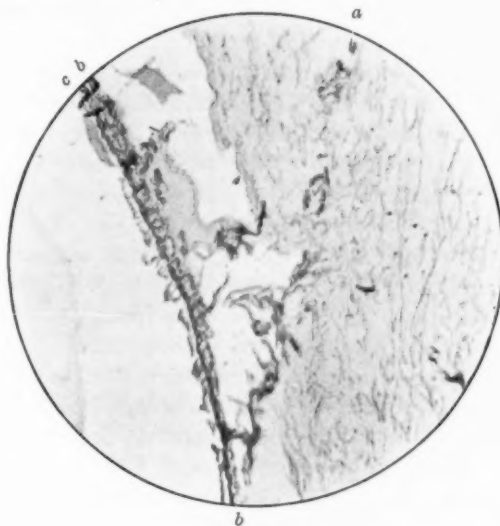


FIG. 3.

Vertical section: *a*, remains of the tooth-band; *b*, remains of the enamel organ showing the three layers described. External epithelium in the form of buds and loops connected with a marked layer of cells (? stratum intermedium) and an inner layer of ameloblasts, *c* (shown well in other parts of the section). Age two weeks.

the last shows numerous buds and loops projecting into the neighbouring connective tissue. The enamel organ undergoes changes as it develops its special function, but with these I shall not deal, although important changes in the external epithelium are described. The stellate reticulum disappears and the inner layer (ameloblasts), with the stratum

intermedium, comes into contact with the outer layer. This is well seen in my earlier sections, where these different strata of epithelial cells consist of an inner layer of columnar cells in contact with the enamel; next to this is a layer which may be more than one cell deep, composed of flattened cells placed end to end (these may correspond to the stratum intermedium), and an outermost layer. The outermost cells are slightly elongated and vary in number, forming irregular layers; they are usually numerous and form slight projections or loops where they are connected. These loops and projections are most marked in the neighbourhood of the remains of the toothband, which can be seen distinctly in early sections. The remnants of this band (in my sections) are connected with the external epithelium towards the upper part of the side on which the permanent tooth germ lies. It is difficult to say how much of these columns is derived from the external epithelium of the enamel organ and how much from the remains of the toothband (or remains of the connecting cord, as it would be here). The cells in the processes projecting from the remains of the enamel organ are rather more rounded or polygonal than those situated directly outside the two layers first described, but their continuity is such that they have obviously been derived from the external epithelium, with the exception just noted. The nuclei in all the cells of this outermost layer are active and deeply stained. Sections cutting this epithelial cap in different parts show that the activity of the external epithelium of the enamel organ in producing buds, varies. Before the complete loss of the stellate reticulum it is very marked at the lower part of the enamel organ, but the most important point to us is the greater activity in the region of the remains of the toothband. At a later period in sections of the tissues lying immediately over the tooth which is just about to erupt the epithelial lining of the sac also varies according to the region cut. In one part, usually over the apex and rather to one side, marked activity with the formation of processes projecting into the surrounding connective tissue is seen, but at other parts the epithelial cells are arranged in layers several cells deep (there may be as many as twenty) and sharply marked off from the overlying tissue. The cells nearest to this tissue are seen to be the most active, whilst those nearest to the tooth are often flattened or appear to be degenerating. The columnar cells (ameloblasts) are not present at this stage in any of my sections. On the outer side of this cap of epithelium the connective tissue forms a very definite zone, and is markedly different from the tissue of a similar character which lies over it. In this definite zone the

connective tissue is very fine, loose and not well stained, whilst the epithelium found there is undergoing degeneration or atrophy. The epithelium exists as small columns and frequently as scattered cells; the cells of the outermost layer of the epithelial cap (external epithelium of the enamel organ) are proliferating, and apparently those cells which pass into this zone as isolated cells rather than in the form of buds are undergoing atrophy. This area can well be described as a "zone of rarefaction," for associated with the changes occurring in the epithelial cells situated there the connective tissue becomes markedly changed in character, being much looser and finer, as described above. This zone of rarefaction is found in all sections of the tissue over the tooth in the stages of eruption. The epithelial cells are all of the polyhedral type; those on the tooth side of the epithelial cap are frequently considerably flattened, whilst those undergoing degeneration or atrophy show badly-stained cells, which are swollen and granular, with indistinct nuclei, but it is not common to find these cells distended with fluid. The zone of rarefaction is well marked off from the tissue overlying it, as the latter is deeply stained.

(2) *The Toothband and the Connecting Bridge.*—We have already seen that there is a more or less direct continuity of epithelial tissue between the surface epithelium and the epithelial cap of the tooth. Also that this epithelium exists in the form of cords or columns, which are tortuous, and that near the surface the arrangement is such as to suggest its persistence in the form of a band. In vertical sections comparison can be made between the different parts of this structure. The cells are almost entirely of the polyhedral type, except when undergoing changes which I am about to describe. The columns of cells are usually seen cut across so as to appear as isolated collections which present a rounded outline. They vary very much in size, often being large enough to be visible to the naked eye, but more numerous smaller collections are also found. The largest masses are found nearest the surface, and it is in these particularly that the cells undergo the change already referred to, although it is also not unfrequently found to be occurring in the smaller collections. In this metamorphosis the central cells proliferate, the outer become flattened and elongated, so that together they form long coiled fibres, a most characteristic appearance. Later, the central cells show marked degeneration, becoming swollen and indistinct; it is probable that the degeneration is complete and of a fluid character, for spaces are found which are never fully occupied by the elongated cells. Where this change occurs all the cells are not

necessarily involved. It is common to find these coils with no active cells present, and only with difficulty can the remains of the nuclei be seen. At times active cells may be found surrounding the coil, or cells which have undergone no change may be situated to one side; the latter is presumably due to the column being cut obliquely, as the mass is usually oval. The coils are more or less spherical, as they are almost circular in section, in whichever plane they are cut. The changes apparently take place at certain points in the columns. Without doubt these fibre-like coils are derived from epithelial cells for they can readily be demonstrated to be so by using "van Gieson" stain, which colours the epithelium yellow and the connective-tissue fibre red. The columns connecting these coils are frequently quite small. The distribution of these coils is of interest; they are found in connexion with the surface epithelium and the remains of the toothband, but not in the region of the tooth-follicle. They are mentioned by Legros and Magitot who speak of them as "globes épidermiques," but they do not give any account of their structure, although they describe their distribution, as also does Malassez. Possibly Robin and Magitot gave a detailed account of them, but I have been unable to obtain a copy of their paper.¹ These coils, when large, apparently open out on the surface; the smaller (especially those deeper in the tissue) appear at times to become flattened out. In transverse sections, what was described as possibly the remains which existed in the form of a band is seen to consist of masses of cells placed side by side, many of them undergoing transformation into epithelial coils. I venture to call these structures "epithelial coils," as the name is suggestive of their appearance; the names which have been used formerly are loosely applied, and are not altogether satisfactory. The degenerative changes which have already been described as occurring in the processes derived from the epithelial cap of the tooth apply also to the cells of the remains of the toothband as the process of eruption takes place.

(3) *Surface Epithelium*.—The surface epithelium shows very little alteration at first. In places it is seen to be thinner. It is also thinner where it lies over a superficial epithelial coil, except where the coil is in contact with it, when it may be considerably thickened. The deeper

¹ In the discussion following this paper these structures were spoken of as the "glands of Serres." On referring to the "Essay" by Serres [11] I believe he probably refers to these, although he does not describe their characteristics, dealing more with their function than with their structure.

layers of cells of the surface epithelium can often be seen surrounding one of the coils. It is difficult to say whether this has originated in the surface epithelium or has become enveloped by it; the former process almost certainly takes place during the later stages, but the coils are not so large as those arising in the upper part of the toothband at an earlier period. Marked changes occur in the surface epithelium as the tooth approaches it. The large quantities of epithelial cells scattered throughout the tissue are partly derived from the two sources already named, but also from the surface epithelium; it is difficult to say how much is



FIG. 4.

Epithelial coils; smaller collections of epithelium can be seen, some of them undergoing change.

contributed by each. The deepest parts of the interpapillary processes at those points where epithelium is present in the subjacent tissues show marked proliferation. These processes may extend to a greater depth, or the epithelium may be of considerable thickness without showing any marked processes, the deeper cells being very active; they may also be seen forming epithelial coils. The proliferation is of such a character that the basement membrane appears to be lost and the epithelial cells appear to be in direct relationship with the connective

tissue. The presence of epithelial coils in the surface epithelium has already been mentioned. These can be seen even to the stage of perforation of the gum by the tooth, but they are not large nor are they so common. When the tooth reaches the surface the epithelial cap comes into contact with the surface epithelium, and sections show a thin layer of epithelial cells over the tooth. These two layers of cells can be seen connected at the margin of the perforation.



FIG. 5.

Epithelial coil opening on the surface.

THE CHANGES OCCURRING IN THE EPITHELIUM AND IN THE CONNECTIVE TISSUES.

These changes have been described to some extent in the above account, as the productive and degenerative or atrophic changes are seen to be taking place at the same time. The latter changes occur particularly in the area immediately outside the external epithelium of the enamel organ or epithelial cap, and this I have called the "zone of rarefaction." It is exceedingly difficult to distinguish exactly what is the nature of the tissues here. I think the term "rarefaction"

perhaps expresses the change better than degeneration or atrophy. When first examined with a low-power objective the tissues seem to be almost entirely composed of a very fine and loose fibro-connective tissue, with several blood-vessels, some collections of epithelium, also rounded spaces, from which it is probable the epithelium has been displaced. On careful examination with high-power objectives the structures are found to be less simple than at first appears. Immediately outside the epithelial cap over the tooth cells can be seen

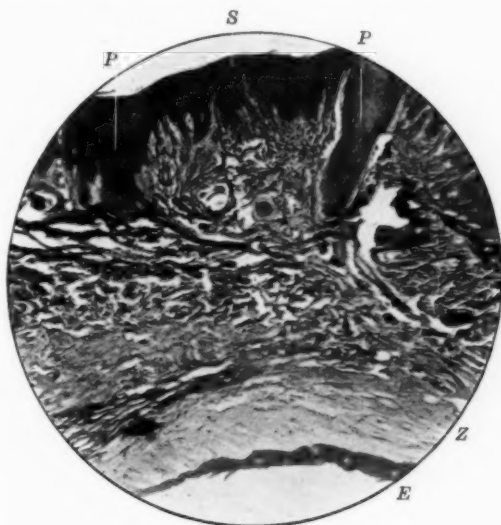


FIG. 6.

Vertical section of cap of tissue over an erupting temporary canine. *E*, epithelial cap lining tooth follicle; *Z*, zone of rarefaction; *S*, surface epithelium with enlarged interpapillary processes, *P*.

which have obviously been derived from it; these cells vary, but many can be seen with a faint outline and badly stained, the distinctness of the nucleus varying with the state of atrophy. The cells frequently have an appearance as though branched; in fact in some parts the reticular character of the tissue appears to be largely due to the branch-like appearance of the altered epithelial cells. It is possible this appearance is due to degeneration of the epithelial cells in the fibro-connective tissue, which is also undergoing atrophy, or perhaps to the disintegration of the cell, the cement substance remaining giving

rise to the reticular appearance. It is an extraordinary fact epithelial cells should be found scattered in this manner in fibro-connective tissue. The connective-tissue cells can also be seen to be undergoing atrophy with the other structures situated in this zone. The nature of this zone can best be studied just previous to the perforation of the gum. In the process of preparation the tissues here are very likely to suffer, and in some of my sections they appear almost as if they had been brushed in order to remove all the cellular structures. It is very probable that the cells would be much more easily removed when under-

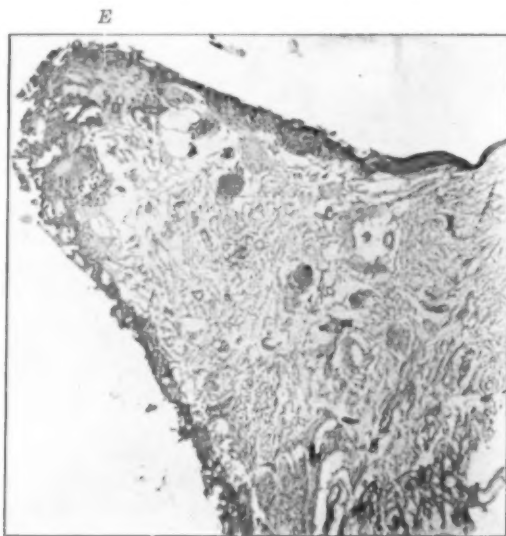
*E*

FIG. 7.

Tongue of tissue between cusps of erupting tooth, showing, *E*, epithelial cap; numerous collections of epithelium can be seen.

going these atrophic changes. Counter-staining with "van Gieson" stain is of some assistance in determining the character of these tissues.

The other change of particular interest to us is the formation of epithelial coils. These have been described already in some degree. They are found in the remains of the toothband and in the surface epithelium. In the early stages they arise chiefly in that part of the toothband which is nearer the surface, and here they are large, presenting a most characteristic appearance, the largest usually being

nearest to the surface, and they are found to be smaller as the tooth is approached. Although they may be seen quite near to the temporary tooth follicle, in connexion with the permanent toothband, they are never found directly connected with the external epithelium of the enamel organ. The epithelial coils which are seen on examination of the surface epithelium are derived from two sources. Some have originated in the surface epithelium itself, whilst others formed primarily in the toothband have become enveloped by it; these open out on the surface. The depression produced by this "opening out"

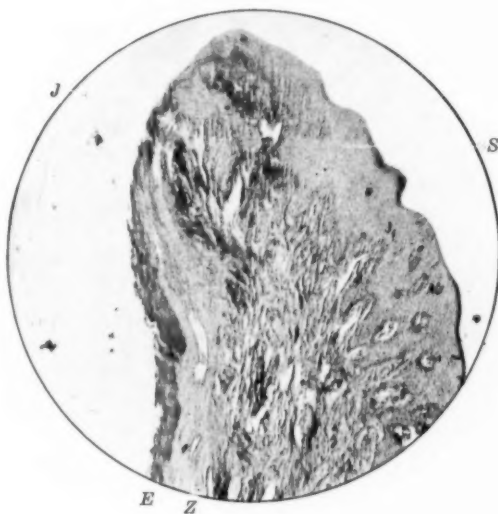


FIG. 8.

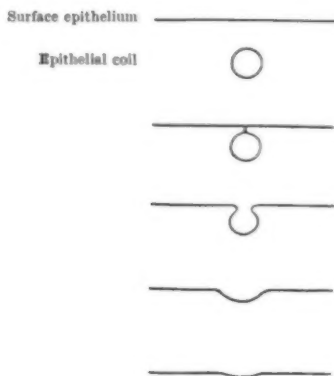
J, junction of *E*, epithelial cap, *S*, surface epithelium, and *Z*, zone of rarefaction.

might possibly be mistaken for a fold in the superficial epithelium, but the exact relationship can only be determined by examining successive sections. The outline of the epithelium surrounding the depression (see fig. 5) should show some indication of interpapillary processes if this were merely a fold. This opening out on the surface is the chief mode of termination of these epithelial coils, but some of the deeper coils appear to be flattened, and it is possible they may be obliterated in this way; but it applies to only quite a small number, and probably never to the larger coils.

THE IMPORTANCE OF THESE CHANGES IN ERUPTION OF THE TEETH.

It is not easy to appreciate fully how the epithelium will assist the tooth in eruption. The chief function I wish to attach to it is that it directs the tooth to its position in the gum. The path of eruption is prepared by the degeneration of the epithelium; the tissues, which are apparently very dense, become loosened and rarefied by the ramifications of the epithelium, and particularly by the changes in the zone of rarefaction. The epithelium probably extends completely from the tooth to the surface; if the continuity be incomplete the epithelial processes can very readily come into relationship with one another.

In the first stages of eruption the epithelium (ameloblast) is in connexion with the tooth, so that the epithelial cap must be carried with



the tooth if it actually moves through the tissues in this stage. It may not do so, for the part played by the epithelial coils may be sufficient to account for the diminution in depth of the overlying tissues. This diminution is brought about by the epithelial coils opening out on the surface, this "opening out" being largely due to the lateral growth in the tissues with the increase in size of the jaw. This opening out will thus lead to a diminution in depth equal to that of the epithelial coil—a considerable amount, as one can see in the vertical sections (*see diagram*). The increase of the tissues in the vertical plane must be remembered. It would be of great value to determine the depth of the tooth below the surface at different ages. The only record I am acquainted with which

might be of value is the atlas of Symington and Rankin [9], but it is insufficient for this purpose. One factor of considerable importance is the growth of the tooth, which must play a part, although it is well known it is quite insufficient to account for the whole process.

In the last stages the epithelium lying *immediately over* the tooth appears to undergo degeneration. I hesitate to state this definitely at present, although I am much inclined to think that it occurs. It is almost impossible to gauge to what extent the cells have been lost during the process of preparation. Should this change occur, it would undoubtedly aid the process of eruption. Clinically, we are all acquainted with the small cystic swelling found at times over an erupting tooth which is described by Tomes. The model which I have here from the museum of the Royal Dental Hospital demonstrates this. I have endeavoured to collect this fluid, but I have not been successful owing to the small amount of fluid present and the difficulty of keeping the child still. It is well known that the cap of tissue overlying the tooth, immediately before it erupts, can be very readily separated, and it is possible to pass a probe between this tissue and the tooth as far as its neck.

It is worthy of note that clinically the first molars are frequently found to articulate immediately the gum is pierced, and in such a case absorption of the gum will play a considerable part in completing the eruption of the tooth, but this factor cannot possibly be of value with regard to the premolars. It is interesting to compare the process of eruption during the development of a hair. "As the young hair reaches in its growth the upper part of the follicle, the central cells which block the neck of the follicle undergo a kind of fatty degeneration" [10] (Quain).

I have called this communication a preliminary note, as I feel that so very much more work has to be done upon the subject, particularly with regard to the permanent teeth, and especially in those cases where the complete healing of the gum has occurred after the removal of the temporary tooth.

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DISCUSSION.

Mr. HOPEWELL-SMITH was sure that the Section much appreciated the most excellent paper to which they had listened. It represented a vast amount of work; a great many sections had been prepared by imbedding in paraffin, which involved a lot of time. The paper showed that a question like that of the eruption of the teeth and many problems which they had to face could be treated by various methods. There might be a histological aspect of the question, but there were other sides—the physiological and the pathological sides. The speaker had given his hearers the interpretations he had placed on examination by the minute anatomy of development. The pathological side of the question was a very important one also, on which they ought to have in the future a paper. He meant one dealing with the causes of non-eruption or partial eruption. In following the lecture that evening it seemed to him that the epithelial coils that Mr. James described were what they had always known as the so-called glands of Serres. Mr. James had pointed out that a probable function of those so-called glands of Serres is the atrophy of cells and the formation of spaces into which the teeth might erupt. But as to whether those epithelial coils existed in the case of teeth which erupted in the ill-formed bone found in the teratomata, or in cases of eruption into the nasal fossæ, one did not know anything at all of that side of the question. Still, behind all there was an eruptive force about which he would like to know the truth. In a way of speaking, he thought it was simply a physiological process. One could not explain it, or reduce it to a mathematical formula or a histological deduction. What was that force? Many theories had been written or

expressed on the subject, but he did not think any were particularly satisfactory. Personally he believed the eruption of the teeth was purely a physiological process, and could not be written down as being especially due to this, that, or the other thing. There was no denying that there is a force behind the eruption of teeth; it was a physiological matter beyond their comprehension, or at all events past any method of interpretation. Mr. James had called his paper "A Preliminary Note"; he (the speaker) hoped that before long he would give the Section a concluding note.

Mr. SYDNEY SPOKES thought they must all admire the energy which had led Mr. James to track his "coils" to their lair in the way he had done. He thought he was to be distinctly congratulated in tracing out that winding pathway, but was hoping they should hear a little from the lecturer as to the formation of that little bag of fluid which was often clinically found preceding the crown of the tooth just underneath the free surface of the mucous membrane. As he understood, Mr. Warwick James in many of his cases had been able to trace the epithelium in continuity from the inside of the dental follicle. It seemed to him (Mr. Spokes) that in that case there was no room for that little bag of membranes, which always seemed to prepare the way by hydrostatic pressure for the advance of the crown in a somewhat analogous way to what the obstetrician felt with his finger in the dilatation of the *os uteri*. Perhaps Mr. Warwick James might in future be able to tell them how the little bag of fluid was formed and where that fluid came from. That might be a little side-issue for an original investigator to track out and give some more information upon.

Mr. J. G. TURNER said that Mr. James mentioned that he made some sections, and perhaps Mr. James thought he had laid too little stress on them. He (Mr. Turner) was rather proud of those sections when he made them. He attached some importance to them because for the first time it was clearly shown that the crown of an erupting tooth was entirely surrounded by a layer of epithelium facing it, more or less cubical epithelium in the sections he cut from an erupting molar—the second temporary molar of a child. He cut those sections because he thought it was possible that epithelial proliferation had something to do with eruption. His idea was that possibly epithelium in some way helped to destroy the tissues in front of the erupting tooth. One found frequently little gland-like masses of epithelium on the coronal side of the erupting tooth, apparently in some way connected with the buds found on the periphery of the enamel organ. But one found that the buds were the more numerous at the growing point of the enamel organ, so that he was rather led to discount the eruptive value of the epithelium proliferating over the tooth. Again, between the actual gum epithelium and those little masses it was not very common to find much epithelium. One found those masses of Serres, but just at the time of eruption they were no more frequent or no more necrotic (so leaving spaces) than quite early in the life of the child. So he rather considered that if there were anything in eruption

due to epithelium it would be by a secretion between the external layer of the enamel organ and the enamel, and consequent pressure in the same way as the bag of membranes to which Mr. Spokes had just alluded, destroying the tissues and allowing the teeth to be pushed up. He was led to think that these things might be correlated by noting that dentigerous cyst was commonest, in fact existed only, in cases of teeth that were liable to obstructed eruption. He had never seen a dentigerous cyst of the first or second permanent molar; it was common with the third and with teeth of succession. He had never seen one yet with an unerupted and unobstructed tooth. The next thing he noted was whether or no a dentigerous cyst was lined with epithelium? Undoubtedly it was in all cases. There always was epithelium, and there nearly always was the appearance of a few of these columns and gland-like masses in the wall outside the epithelium. The little cysts that were found over erupting teeth were also lined with epithelium, sometimes very thick and sometimes almost necrotic and scarcely staining. So that one looked at a dentigerous cyst as an effort of nature to remove the obstruction to eruption; and one was inclined to think that perhaps normal eruption was connected with something at one end of the scale of dentigerous cyst. Sir John Tomes had already suggested that the fluid that was let out when they cut those small cysts over an erupting tooth might have something to do with the normal process of eruption. Still, there was a difficulty left; because lots of teeth erupted that were covered with cementum; and there was a specimen that Mr. Smale gave to the Society of a dentigerous cyst of a Cape buffalo, caused by an obstruction of an odontome of the first molar, containing the second and third molars—both teeth being covered by cementum. Specimens were wanted to show whether those dentigerous cysts were lined with cementum. Also specimens were wanted to show what was the relation of the cement organ in the normal case of eruption of cement-covered teeth to the epithelium of the enamel organ. He had just got a small lamb's head—one of his own that unfortunately died—and he was going to try and get some sections, and if Mr. James cared to make the sections he would be very pleased. In these cases the bag-of-membranes theory could not apparently hold water, but the road for eruption might still be prepared by proliferating epithelium.

The PRESIDENT (Mr. Leonard Matheson) asked Mr. James whether he was right in supposing that the lecturer's wish was to convey his opinion that the eruption of the teeth was affected more than had hitherto been supposed by the epithelial cells that were found connecting the region of the erupting tooth with the surface?

Mr. WARWICK JAMES, in reply to the President, said that he had a summary at the end of what he thought were really the factors in regard to eruption. He believed one was the epithelial coils, or glands of Serres. Those, he thought, played a part in diminishing the depth of tissue over the tooth. Either they became flattened out—generally they were small if they did so—or

they actually opened out and became continuous with the surface epithelium. The other factor, he thought, was the degeneration occurring in the layer situated outside the external epithelium. One found degenerating epithelium in that particular area. If there were an erupting force—Mr. Turner suggested bone growth; Mr. Hopewell-Smith suggested physiological process—it must be a force of some kind, and normal growth of the surrounding tissues appealed to him (the lecturer) more than anything else. The tooth passes up into the external area which is atrophied, so the tooth could advance with its own growth partly, and partly with the growth of the surrounding tissue; but this is mainly theoretical. The chief point of the paper he wanted to make was that it was the directing factor in bringing a tooth to the surface. If one considered the large number of teeth erupting—the enormous number—and that they always came more or less to the same point in the gum, there must be some directing factor, and he thought it was the epithelium which persisted. The columns of epithelium were not single, but multiple, so that there was some variation possible in the route to be taken. He had intended to say something about the pathological conditions, but thought it was better to leave them alone rather than just to mention them. Certain pathological conditions occurring in the jaw indicated that degeneration could occur in those epithelial masses, and that they very frequently did so. With regard to the molars, he was very interested to hear of the second and third permanent molar in the Cape buffalo being situated in the same cyst. Because, if one considered the toothband in the molar region—taking the second temporary tooth and so on, ending up with the third permanent molar—as they erupt they come up one behind the other, a point of very great importance in determining the position of these teeth. If degeneration occurred in that band, then it would be quite easy for the teeth to be situated in the same cyst. He had not worked at the permanent teeth really; he had a few sections. He laid stress on the fact that the gubernaculum dentis contained epithelium, because he thought the point he was putting forward would be useless if it did not also apply to the permanent dentition. With regard to Mr. Hopewell-Smith's remark as to aberrant teeth—not erupting, but passing in different directions—he thought it was quite possible, by the growth from the external epithelium of the processes which passed outwards and anastomosed. It might be possible for eruption to take place in that way, although there might be no other aiding epithelium. They knew, as a rule, that those teeth which erupted in abnormal positions were late in erupting, and it would very likely take a long time, supposing that to be the explanation. With regard to the glands of Serres, he tried to find the original paper written by Serres, but had only just found the reference to it. He might have very well asked what would be the generally accepted condition of the glands of Serres; but they were so differently described that it was difficult to make out what was meant by a gland of Serres. He did not know exactly that it corresponded to the epithelial coils. He knew the epithelial coils had been described before, as long ago as 1873, by Legros and Magitot. With regard to the bag of fluid, he really had something to say about that too. The internal

epithelium and the external epithelium were in contact up to three months. He found them in nearly all his sections of three months, and even later. But as to the character of the external epithelium, he could not ascertain where it was changed, at what stage exactly they obtained this number of flattened cells or polyhedral cells which Mr. Turner spoke of as cubical. They were several layers deep. It was different from the external epithelium in younger subjects when the internal epithelium and external epithelium were in contact, but later on there appeared to be degeneration occurring on the tooth side of this layer. It was possible that the fluid was formed there by degeneration of the inner side of the epithelial cap, so that they had really degeneration going on on both sides of that layer. It could not continue for a very great length of time.

Odontological Section.

June 28, 1909.

Mr. LEONARD MATHESON, President of the Section, in the Chair.

Radicular Aberrations.

By H. PERCY PICKERILL, M.B., L.D.S.¹

UNDER this title are described specimens which have been recently added to the Dental Museum of this University, donated, many of them, by dentists practising in various parts of the Dominion. The word "aberrations" is used in its widest sense, and some pathological conditions are included; nevertheless, the specimens include four odontomata undoubtedly true "aberrations."

SPECIMEN I.—ABNORMAL LENGTH OF ROOTS.

History: These teeth were removed from the mouth of a girl, aged 19, of rather fragile appearance, with no apparent abnormal development of the jaws, as may be judged from the photograph. The teeth, besides being of excessive length, were exceedingly firmly fixed in the jaws and gave rise to considerable difficulty in extraction.

Macroscopical appearance: Fig. 1 shows two of the teeth—the canine and first premolar from the left maxilla. The canine measures 4.5 cm. in length, and 9 mm. in diameter at the widest part of the root; the crown is not abnormally large, and measures 1 cm. long and 7 mm. wide. Two slight peculiarities are to be noticed about the crown: (1) it has the appearance of and is shaped like a right-sided tooth; (2) the enamel, instead of ending abruptly in the usual manner,

¹ From the University of Otago.

gradually shelves off and becomes lost in an irregular manner upon the root. The apical foramen is still open, measuring 4 mm. by 2 mm.; its edges are sharp and irregular, and it has every appearance of development being still in progress. A naked-eye examination of the tooth as a whole gives the impression that a tooth normal in shape and size had started to be formed, and then, shortly after the completion of the crown, something had occurred to stimulate development excessively. The tooth also appears as if a considerable amount of productive periodontitis had occurred around its circumference in the middle part of

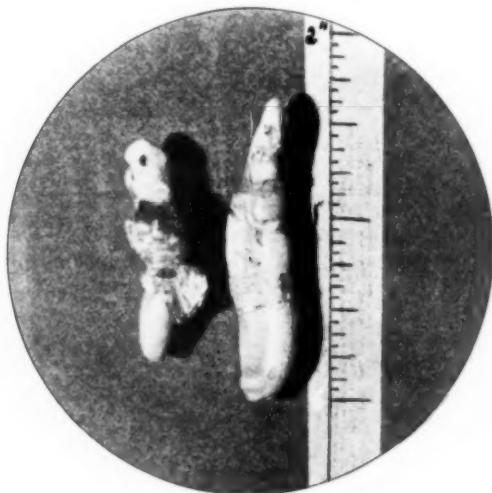


FIG. 1.

Photograph after removal of sections for examination.

the root. Measurement of the tooth against the face of the patient after extraction proved that the apex of the tooth must have reached to within $\frac{1}{4}$ in. of the inner angle of the orbit. The premolar measures 3 cm. in length and 1 cm. in diameter at the widest part of the root; it is seen that there is a piece of alveolus adherent to the mesial and buccal aspect of the root. The foramen is also open, and measures 3 mm. across by 2 mm.; here, too, development appears to be incomplete.

Microscopical appearance: Contrary to expectations, the whole of the tissue in the canine is normal, as seen in a section through the widest diameter. As will be seen, there has been no productive periodontitis. The cement exists in a thin, almost hyaline layer; the dentine, also, is quite normal. Thus it is quite evident that the whole of the tooth was formed centripetally, and its peculiar shape must have arisen through an almost sudden enlargement of the dentine papilla. A point of minor importance is the irregular lateral grouping of the dentinal tubes near their termination in the pulp canal; this would seem



FIG. 2. ($\times 40$.)

to indicate that development had proceeded up to a certain point and then had become, from some cause, hastened and irregular. Fig. 2 is a transverse section through the premolar and the adherent alveolus, prepared by grinding. Here, again, there is marked absence of any signs of productive periodontitis, although the periodontal membrane is obliterated and the alveolus is apparently ankylosed to the tooth.

It is possible that this case is to be regarded as an attempt to produce teeth of persistent growth in the human being.

SPECIMEN II.—RADICULAR ABSORPTION OF PERMANENT TEETH.

Fig. 3 shows two central incisors and one upper molar, extracted from a woman aged 30. All the history obtainable was that there was constant "neuralgia" and that all the teeth were the same as the three shown here.

FIG. 3. ($\times 2$)FIG. 4. ($\times 10$.)

Macroscopical appearance: In length the roots of the central incisors measure 7 mm., the crowns 12 mm.; the roots of the molar 8 mm. and the crown 8 mm. The crowns of the teeth are perfectly normal, there is no caries in the incisors, and only a very small crown cavity in the molar. The roots terminate abruptly—the incisors in a broad, rough

cone, and the molar in an irregular, horizontal surface at the point where the roots should normally have begun to bifurcate. The apical foramina are either invisible or very minute.

Microscopical examination: Fig. 4 shows a section taken vertically through the upper horizontal surface of the molar. The upper border is irregular in outline, and the dentine, which is normal in structure, reaches to within a short distance of it; in one or two places, however, the course of the tubes is abruptly terminated at this edge. The pulp canals are sealed by the presence of adventitious dentine, which in the lower parts is fibrillar, but further up the canals is coarsely granular

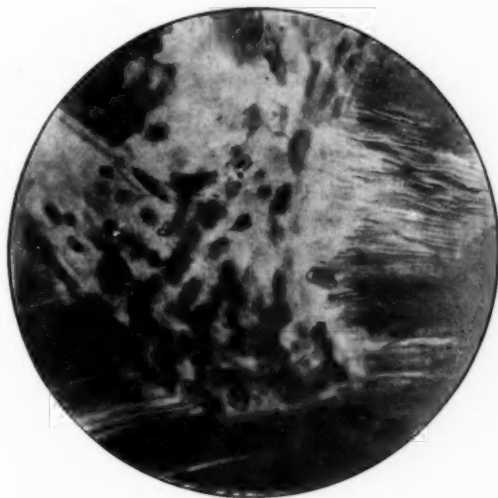


FIG. 5. ($\times 200$.)

with large uncalcified spaces. It will be observed that the dentinal tubes end abruptly and that the dentinal margin shows numerous concavities resembling the faveolæ of Howship seen in the physiological absorption of temporary teeth; filling in these concavities, and extending for some distance beyond, is an indefinite osseous material, which is neither cement, dentine, nor bone—it has no definite structure, contains no true lacunæ or canaliculi, and appears to be irregularly calcified. The history of these teeth, as read from these sections, would appear to be that for some reason absorption of a considerable portion of the root has taken place, a reaction on the part of the pulp has occurred, and

adventitious dentine has been thrown out as a bar to the advance of the osteoclasts. The stimulus for absorption having subsided, repair took place in a limited fashion by the rapid deposit of a calcific material. In the matrix of the material filling up one of the larger faveolæ (fig. 5) there appear to be imbedded the remains of large cells such as are osteoclasts. Fig. 5 resembles very much a section of an "absorbent organ" *in situ*.

The neuralgia complained of might very possibly have been due to pressure on nervous elements by the reparative tissue.

SPECIMEN III.—BILATERAL RADICULAR DENTOMATA.

A photograph of two first upper molars, which were extracted with difficulty in an orthodontia case, is depicted in fig. 6. There are no

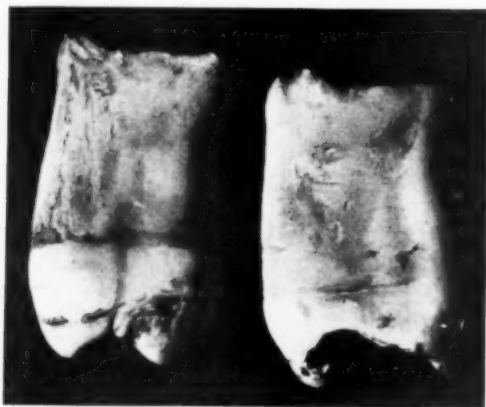


FIG. 6. ($\times 2\frac{1}{2}$.)

separate roots. The teeth are cuboidal, or bale-shaped—*i.e.*, there is a longitudinal groove running down each side. The crowns show large, occlusal cavities, but are otherwise normal. The apices, instead of being conical, are large, uneven quadrilateral surfaces; in both teeth they are practically the same size, and measure 11 cm. mesio-distally and 12 cm. bucco-palatally. From macroscopical appearance only one would think that four roots had spread out, become fused, and the centre filled in with a plug of cement. Microscopical examination shows that this is



FIG. 7. ($\times 8$.)



FIG. 8. ($\times 8$.)

Horizontal section just above the cervix.

not so (fig. 7) ; the whole tissue is perfectly normal dentine ; the only thing abnormal is the manner of its distribution ; instead of there being three or four root-canals there exists only one large central quadrilateral shaped cavity, surrounded by a circular and parallel wall of dentine (*see* figs. 8 and 9). This aberration is, in the opinion of the writer, sufficient to justify the above classification, and constitutes, perhaps, the simplest form of odontome.



FIG. 9. ($\times 2$.)

SPECIMEN IV.—RADICULAR ODONTOME.

Fig. 10 represents a model of the mouth of the patient, a girl aged 13. It is observed that the left lateral incisor is peg-shaped and is markedly procumbent. On the palatal side of this tooth is a hole representing an aperture in the muco-periosteum, 6 mm. in diameter. This aperture led upwards into a cavity whose walls were smooth and hard, the internal diameter of which was apparently about twice as large as the orifice. On the labial aspect there was a hard bluish swelling lying a little posterior to the usual position of the lateral incisor root (fig. 10). The peg-shaped tooth was evidently connected with the bony swelling, for on palpation slight movement was obviously transmitted from one to the other. A diagnosis of radicular odontome was made, but unfortunately, before its removal could be arranged, the patient broke and swallowed the peg-shaped crown ; the remainder was,

however, removed by reflecting the mucous membrane and excising the alveolus. Fig. 11 gives some idea of the shape of the fragment removed; it resembles the pointed end of a filbert-nut shell, and measures 1.2 cm. in length and 8 mm. in width; it is thickest at the apex, and gradually tapers to the free edge, which is obviously carious.

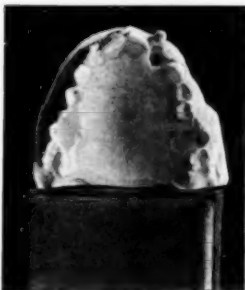


FIG. 10.

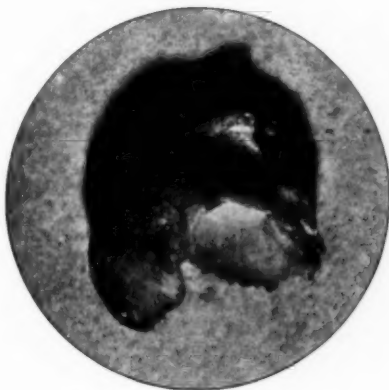


FIG. 11. ($\times 4$.)

At the apex of the tumour a small foramen exists, on either side of which were two small masses of granulation tissue; the outer surface is quite smooth and white, the inner a little rougher and discoloured.

Microscopical examination: A vertical section (fig. 12) shows that the whole of the tissue is normal dentine and cement. On the inner

surface, for about two-thirds of the distance between the free margin and the apical foramen, the surface is irregular—caries has evidently destroyed part of the dentine; for the remaining one-third, however, the dentinal tubes end as they normally do in a pulp cavity, and have no appearance of having been destroyed by caries. If this be so, then there probably was a large central ovoid pulp cavity, surrounded by a shell of dentine, which was continuous with the peg-shaped incisor.

There is a considerable resemblance between this specimen and the last one. They might all, possibly, be described as radicular dentomata, since they are all "aberrations of the dentine papilla after the crown has

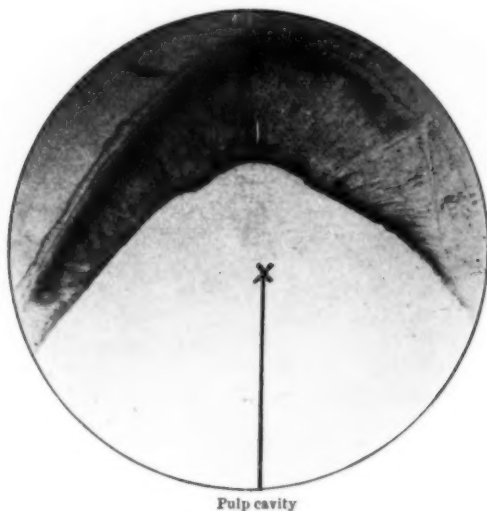


FIG. 13. ($\times 10$.)

developed." If this term does not sufficiently specialize this class of tumour they might be termed "medullary dentomata." The special feature of both seems to be the dilatation of the pulp cavity, with hypertrophy and aberrant development of the dentinal walls.

SPECIMEN V.—COMPOSITE ODONTOME.

This is probably an aberration of the tooth germ which should have formed a third upper molar. As may be seen by fig. 13, it is a solid figure-of-eight-shaped mass adherent to the anterior root of an



FIG. 13. ($\times 2$.)

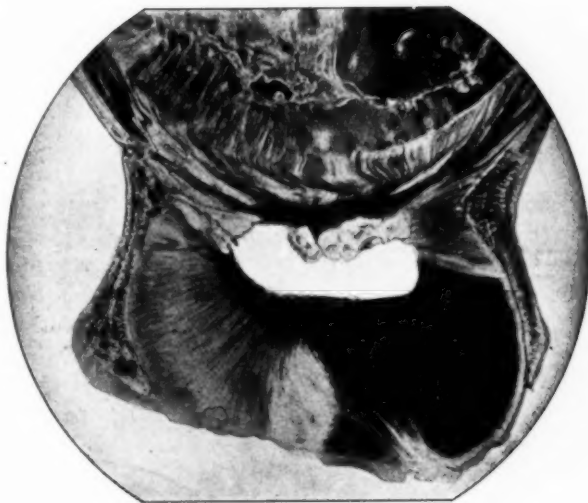


FIG. 14. ($\times 9$.)

upper molar and apparently replacing the posterior root. It measures in length 1.5 cm., in width at its greatest diameter 1 cm., and at its narrowest diameter 7 mm. The external appearance is smooth and yellowish like normal cement; there is not the slightest indication of enamel.

Microscopical examination: A vertical section through its centre shows the whole tumour to be solid, except for the small area in the centre just below the neck. The smaller bulb of the tumour resembles the crown of a normal tooth in shape (fig. 14). There is a central pulp cavity surrounded by normal dentine, the tubes of which converge from the periphery to open in the cavity; but instead of the dentine being covered by enamel, there is an incomplete covering of thick lacunated cement. The dentine is moreover radicular in character—*i.e.*, it terminates in numerous fine dendritic processes in a granular layer; there are no interglobular spaces at all in this portion. In the upper part of the pulp cavity there are some nodules of "adventitious dentine." This has a peculiar structure; for the most part it is hyaline or finely granular, but scattered through are numerous exceedingly fine canaliculi, ramifying in all directions, not communicating with each other and having many fine dendritic processes. These canaliculi do not appear to have any connexion with the pulp cavity, nor are lacunæ present. A figure gives but a very inadequate idea of these processes, since they do not lie on the same plane and only a small portion of each is in focus at the same time. The upper and larger bulb of the tumour, for purposes of description, may be divided into a central mass and three concentric zones (figs. 14 and 15). The outer zone consists of a layer of cement (much thinner than that on the lower part of the tumour), containing lacunæ and canaliculi in moderate numbers. The middle zone is one of fine tubed dentine, the tubes running in a direction parallel to the surface and downwards towards the pulp cavity. The inner zone consists of fine tubed dentine, the tubes of which run centrifugally (*i.e.*, at right angles to the surface). They arise at the inner boundary of the zone among dense interglobular spaces and become continuous with the tubes of the middle zone by an abrupt right-angular bend. The interglobular spaces are exceedingly numerous and extend in some places throughout the entire zone, they are also excessively large, in places resembling the liquefaction foci of caries. Fig. 16 shows one of these large uncalcified spaces, which has fortunately not become filled in with debris. In it may be seen calcoglobular masses in all stages of development—of complete and incomplete fusion. The central

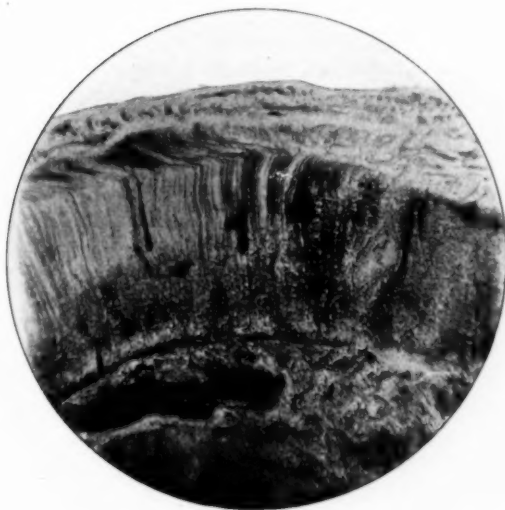


FIG. 15. ($\times 50$.)



Fig. 16. ($\times 250$.)

mass consists for the most part of indefinite osseous tissue irregularly calcified. It resembles bone more than any other tissue, but there are no canals and no true lacunæ and canaliculi, merely irregular uncalcified spaces. The most interesting point about the whole tumour is that projecting into this central mass from several points on its periphery are small masses of enamel. Fig. 17 represents one of these. The structure of the enamel is poor; the prisms are not distinct and do not run for the most part in a definite direction, but appear to wind about in various planes. The outer parts of the nodules nearest the osseous tissue are "pigmented," whilst that abutting on the inner zone of dentine is more granular and penetrated by enamel spindles, the latter being, as usual, in

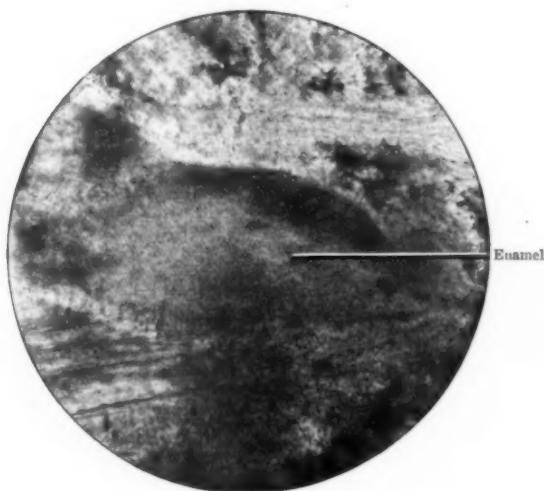


FIG. 17. ($\times 150$.)

direct communication with the dentinal tubes. It is noticeable, too, that where the enamel nodules are present the dentine of the inner zone is much more normal in structure and contains few, if any, interglobular spaces. By what means portion of the enamel organ could have become included in the centre of such a tumour, or a "crown" formed in normal shape without any deposit of enamel or evidence of enamel organ, is not at all clear. For if ameloblasts had been present during the development of the crown, but had not calcified, some remains or

evidence of their presence might have been expected between the dentine and the cap of cement—but none exists.

SPECIMEN VI.—OSTEOMA OF THE PERIODONTAL MEMBRANE.

Fig. 18 represents the microscopical appearance of the specimen. To the posterior aspect of the apex of a rather small third molar is attached a spherical mass of dense bone measuring 5 mm. by 8 mm. The surface is uneven but smooth, and of a dull white colour. The crown of the tooth presents a carious cavity on its mesial aspect.



FIG. 18. (x 2.)

Microscopical examination shows the spherical mass to be composed of compact bone, not quite normal in structure but showing (a little indefinitely) the formation of Haversian systems (fig. 19). Lacunæ and canaliculi are very numerous and show grouping around central canals, some of which are cut transversely and some longitudinally, but it does not in the least resemble alveolar bone. There is a layer of normal cement covering the root. This is succeeded by several strata of hyperplastic cement, which merges imperceptibly into the bone. There is no sign of any absorption having taken place. If this were a case of ankylosis of the tooth to the bone of the jaw one would expect marked evidence of previous acute inflammation, but there is none. The whole character of the tumour, too, is one of slow, definite, and

compact growth, resembling that of any other true osteoma; the hyperplasia of the cement was quite probably caused by the irritation of the neoplasm.

SPECIMEN VII.—FUSION AND INTERLOCKING OF MAXILLARY MOLARS.

The last specimen is an ankylosis of the left upper second and third molars. The third molar lies horizontally, the roots being embraced by, and absolutely fused with, those of the second molar.



FIG. 19. ($\times 12$.)

Each tooth shows signs of three roots, but without spaces between them. The second molar is worn by attrition, but there is no caries. The third molar is unworn and evidently has never been erupted.

Microscopical examination: In a section taken vertically through the palatal root of the second molar and transversely across the roots of the third molar it is seen that fusion has occurred through the cement alone, which is exceedingly hyperplastic. All three roots of the third molar are fused through the dentine alone, and at the point where the palatal root joins one of the buccal roots an island of

imperfectly formed cement is enclosed in the dentine. This appears at first like a second canal obliterated by calcareous degeneration of its pulp. On closer examination, however, the dental tubes are seen to begin in the normal granular layer of Tomes in the centre and to radiate outwards.

In conclusion, the writer wishes to record his indebtedness to Mr. S. H. Rawson for his assistance in the preparation of the lantern slides.

All the sections were ground hard and are unstained.

Mr. DOUGLAS P. GABELL thought the only specimen of Mr. Pickerill's which was questionable was the one showing osteomata of the root. The author described it as having no signs of inflammation in the tooth; but in the photograph shown there was a very large granular layer of Tomes, which was usually found associated with chronic inflammation of the periosteum externally. The differentiation from cementum of the tumour, shown in the slide, seemed to be so very small that he did not know why the author should claim that it was an osteoma, and not entirely an exostosis, although an exceedingly large one.

A New Interdental Splint.

By J. LEWIN PAYNE, M.R.C.S., L.D.S.

THE communication which I am bringing before you this evening consists of an account of a simple interdental splint that I have found useful in the treatment of fractures of the jaw and in those cases in which some appliance is required for the purpose of retaining the mandible in its relative position of occlusion after operation. The two considerations which most concern the surgeon, or the dental surgeon, who has charge of these cases are the relationship of the fragments to their normal position and the cleanliness of the mouth. The first point has a bearing upon the second, for so long as the fragments remain unstable it is difficult to keep the mouth clean, and undue mobility promotes suppuration. In any case the tendency to sepsis is considerable. This may be attributed (1) to the fact that there is usually direct communication between the injured bone and the cavity of the mouth; (2) to the difficulty of retaining the fragments in position;

(3) to the form of the splint commonly employed; and (4) it may well be added that the type of patient in whom fracture of the jaw most commonly occurs rarely understands the elements of the hygiene of the mouth, or, if he does understand, he fails to practise them.

A splint which encloses the teeth completely must necessarily interfere with the hygiene of the mouth, and whilst it cannot be said that it is yet possible to discard the vulcanite and the metal-plate splints entirely, I do think that the cradle splint, about to be described, can, in a very large number of cases, be used instead of them with advantage, not only in regard to the cleanliness of the mouth and the comfort of the patient, but also in obtaining the required stability of the ends of the fractured bone. It is necessary to explain first of all that this splint, in its original form, is only suitable in cases in which there are several teeth present; there should be not fewer than four or five in each jaw, and their implantation should be firm.

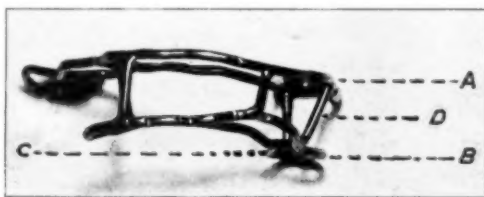


FIG. 1.
The cradle splint.

The method of constructing this splint is as follows: When the impressions of the upper and lower jaws have been obtained and cast in plaster of paris, the model of the fractured jaw is sawn apart if it shows displacement of the fragments, articulated with the opposing model, and is then reunited in the usual way. Metal dies of both the upper and lower models are next made, and a silver-wire framework is fitted to each of them close to the necks of the teeth on the lingual and labial aspects, also as far round the arch as it is convenient to carry the splint. A very tight fitting cradle is neither necessary nor desirable. This is perhaps fortunate, since, as is well known, in many, if not in most, cases it is difficult to secure entirely accurate impressions owing to the conditions which prevail. Silver wire, size 4 or 5, is used; it is sufficiently soft to bend with pliers when the splint is being adjusted to the mouth, and yet

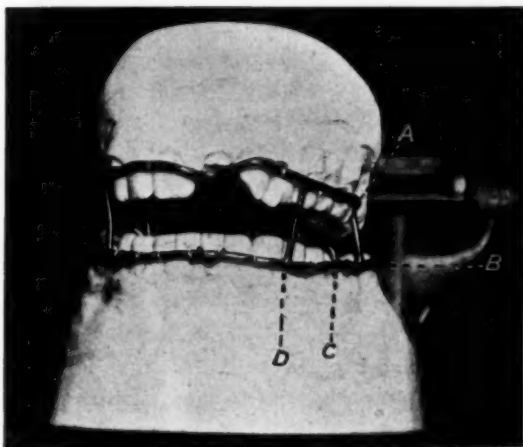


FIG. 2.

The cradle splint adapted to models of a case of fractured mandible.

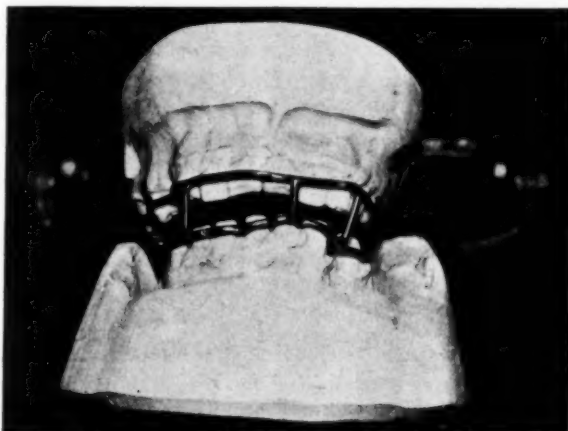


FIG. 3.

The cradle splint in use for a case where the mandible has been removed behind the right canine for malignant growth.

strong enough to hold the fragments firmly in position when the cradle is completed. The ends of each half of the framework are soldered together, and at this point they resemble a pair of Hammond wire splints, which fit the teeth of the upper and lower jaws (A and B). The next step is to strengthen the arches by soldering bars (C) to them at suitable intervals; these bars unite the labial and lingual sides. It is convenient to have two or three such bars in each arch; they can be placed at intervals between the cusps of the teeth, or in spaces left by teeth which have been extracted. These bars not only help to strengthen the splint, but they also give it firm grip and prevent it slipping on to the gum. The plaster models are then mounted in the position of correct occlusion on a Gritman or other anatomical articulator, and when the two wire arches have been adjusted to them the bite is raised about $\frac{1}{4}$ in., or to such a height as will conveniently allow of the introduction of food when they are in position, but not so high as to interfere with the insertion of the splint. The two arches are then united by vertical rods (D) of silver wire, two or three of which are soldered on the labial and lingual sides. This done, the cradle is complete, and after it has been trimmed up, polished and gilded, it is ready for insertion. I make a practice of gilding the cradle in order to prevent the discoloration which otherwise occurs when a silver appliance is worn in the mouth.

In fractures of the jaw where the displacement is small and the muscular pull is not strong the splint will keep the fragments well together without aid, but in most cases it is better to attach it firmly to the teeth by means of wire ligatures wound about it in figure-of-eight fashion, as employed for Hammond's splint. Three or four ligatures applied to each jaw will usually hold the parts in good apposition.

The advantages claimed for the cradle splint are these:—

(1) The teeth are retained in their normal position of occlusion, so that, even if the fracture is behind the last tooth, the cradle is still available.

(2) It grips displaced fragments firmly and retains them in position.

(3) It is clean in itself.

(4) It does not prevent the cleaning of the teeth while it is in use.

(5) The condition of the jaw, and the progress which is being made, can be observed without interfering with the splint.

In conclusion, I would also urge its use in those cases in which, either owing to a malignant growth or other serious cause, a section of the mandible has been removed. The insertion of the cradle splint a

day or so after operation not only controls the tendency to deformity, but it also hastens the healing of the wound, and the patient finds it a source of considerable comfort. The cradle splint is so simple, both in form and construction, that I hope it may be considered worthy of adoption by the members of this Section. I have never seen such a splint previously described, and, as it has proved most successful in the cases in which it has been employed, I have ventured to bring it to your notice this evening.

DISCUSSION.

The PRESIDENT (Mr. Leonard Matheson) thought the very simplicity of the apparatus made it exceedingly attractive, especially as it seemed to be adaptable to very many cases. It would be interesting to hear whether any of the Fellows had used a similar form of splint. The author was to be congratulated on having hit upon a very valuable means of dealing with cases which were often troublesome.

Mr. NORTHCROFT inquired how long the author found it necessary to leave the splint *in situ* in cases of removal, for instance, of half the mandible. It seemed to him that while a splint of the kind described might be successful for a time, the period that elapsed while the tissue was contracting was so great that the patient would have to wear the splint for a very long time for it to be of any ultimate gain. He would also like to know if the gauge of silver wire that Mr. Payne used was that used by the Depots, which was a purely arbitrary one. He believed No. 5 was 2 mm. in diameter.

Mr. C. F. RILOT said the splint handed round seemed to him to have no rest over the teeth, and asked the author how he prevented it being forced up on the gum margins from the pressure of the jaws.

Mr. J. LEWIN PAYNE, in reply to Mr. Northcroft, said the gauge of the wire he mentioned was the somewhat rough one which was given by the Depots. With regard to the employment of the cradle splint in those cases where a section of the jaw had been removed by operation, it was necessary to retain the splint in position for about three months, and upon its removal another appliance should be inserted, one which, whilst affording the power of mastication, should enable the patient to retain the jaw in its normal relationship to the upper teeth. The patient was not likely to be able to masticate in less than three months after the operation, but the insertion of the cradle splint described would not seriously interfere with the taking of food. Those who had had cases of fracture of the jaw to deal with were familiar with the wonderful amount and variety of food which patients were able to take, even when wearing a Gunning splint; if the food were only well chopped up, they could, after a time, take almost anything in the way of nourishment. Mr. Rilot had inquired how the pressure on the gum was

prevented. Ordinarily one employed bars, marked C on the diagram, running across in between the cusps of the teeth. As the bite was raised there was plenty of room for the bars, which by resting between the cusps held the cradle in position. It was not always necessary to use these, however, for the cradle could be fixed in the same way as the Hammond splint with figure-of-eight wire ligatures, as suggested in the paper; but for cases where there was likely to be any pressure on the gum the crossbars C should be employed.

An Epithelial Odontome.

By WARWICK JAMES, F.R.C.S., L.D.S., and
J. GRAHAM FORBES, M.D.

A SMALL girl, aged $4\frac{1}{2}$, had a swelling situated in the anterior part of the right side of her mandible. She was quite healthy, and no history of local injury could be obtained; the temporary incisors and canine of the right side were missing; from the mother's account they had become loose and dropped out, they were not carious. The swelling was just noticeable externally, and beyond the slight inconvenience of its presence there was nothing of which the patient complained. The tumour had gradually increased in size since the mother had noticed it a few weeks previously.

On examination a rounded swelling was present in the mandible in a position corresponding to that of the teeth lost. It extended from the first temporary molar of the right side to the symphysis, just crossing the middle line; the left central incisor was pushed over slightly to the left. The size and shape of the tumour can be well seen in the model. The swelling—somewhat globular in outline, suggesting a fluid character—was situated in the alveolar portion of the bone, the latter existing as a thin bony capsule over the tumour, yielding on pressure the characteristic "parchment crackling." Fluctuation could be obtained where the bony capsule was deficient in front of the temporary molar. The colour and appearance of the gum were normal, the points from which the teeth had been lost were still indicated, a little granulation tissue being present where the right canine had been situated.

The removal of the tumour was effected as follows: Two incisions were made in the line of the gum ridge to include a fusiform portion of

the overlying tissues and carried down to the capsule. The thin layer of bone was easily cut through, and by using a periosteal elevator the tumour was completely shelled out without difficulty. When separated from the surrounding tissues it proved to be an encapsuled cyst, oval



FIG. 1.

Photograph of the tumour *in situ*.



FIG. 2.

Model showing the size and position of the tumour. The appearance of the molar teeth is due to a defective impression and not to caries

in shape, measuring 12.8 mm. by 8.5 mm. On incision through the capsule a small quantity of fluid escaped, but the bulk of the contents was composed of loose granular material. The whole of the excised cyst, including the overlying and adherent portion of gum, was carefully examined in section under the microscope.

Microscopical appearance: The capsule, measuring 2 mm. in thickness, was composed of layers of dense fibrous tissue enclosing a papilliferous cyst, and continuous on each side with the fibrous tissue of the papillary layer of the gum which formed an overlying cap. Included in the fibrous tissue were seen irregular trabeculae of bone. Attached by narrow pedicles to the cyst wall occurred long slender irregular papillary processes covered with flattened or oval epithelial cells, which also extended down the fine connecting stalk to the cyst wall (fig. 4). These processes were seen in various planes; tangential section



FIG. 3.

Skiagram taken previous to removal of the tumour. (The negative was placed beneath the chin, so that here the tumour appears to be on the left side.)

through the covering showed an irregular mass of epithelial cells, round, oval, or flattened into a fusiform shape and occasionally grouped in concentric layers. Longitudinal section showed the pedicle to carry a capillary vessel, which in places was dilated into a blood-space of irregular size and shape (fig. 5). Transverse section succeeded in demonstrating an alveolar arrangement of epithelium in the form of rounded alveoli lined by cubical or columnar cells, and surrounded by layers of flattened, stellate or oval cells. Many of the papillary outgrowths into the cyst differed in shape from those just described, and appeared as short epithelial tufts growing from the lining wall. Films prepared from the more fluid contents of the cyst showed numerous stellate cells and degenerated epithelium occurring free or in conglomerate masses.

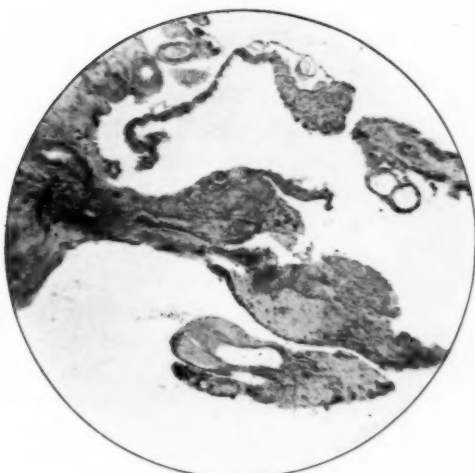


FIG. 4.

Section showing a papillary process.

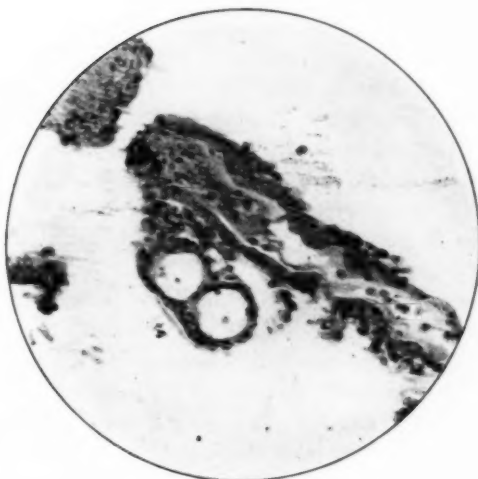


FIG. 5.

Higher magnification of part of the section shown in fig. 4.

The more solid granular particles, which escaped from the cyst on incision, were seen to be formed of epithelial cells grouped together in concentric masses, and in alveoli loosely united by stellate connective-tissue cells (fig. 8). The alveoli were lined by cubical or definitely columnar epithelium and surrounded by layers of small flattened or oval cells, apparently epithelial in origin (figs. 6 and 7). It appeared probable that these structures were the remains of the papillary processes originally growing from the inner wall of the cyst, but which in course of growth had become detached and formed free collections of epithelium.

In addition to the papillary tufts and processes attached to the inner wall of the cyst and forming the bulk of the tumour, some sections showed proliferation of the surface epithelium of the gum and production of irregular downgrowths of the stratum Malpighii coming into close contact with the more superficial part of the cyst wall (fig. 9). Sections taken in the periphery of the tumour showed that these surface proliferations extended through the fibrous tissue overlying the cyst to reach the papillæ derived from the epithelial upgrowths of the tumour, so that there appeared an intermingling of the epithelial structures growing down from the surface and of those growing up from the deeper tissues belonging to the cyst.

In attempting an explanation of the origin of the tumour it is necessary to consider briefly the structure and changes in the enamel organ. Previous to calcification the enamel organ consists of an internal epithelium, consisting of long, delicate columnar cells; the outer part consists of the external epithelium, which is composed of oval or cubical cells; between these is the enamel pulp or stellate reticulum. The external epithelium of the enamel organ becomes considerably altered by proliferation of its cells, forming buds projecting into the overlying tissue; also processes carrying blood-vessels are said to project inwards and to come into direct contact with the enamel pulp. In the normal process of development the stellate reticulum disappears and the internal epithelium comes into contact with the external epithelium. It is also of interest that the surface epithelium shows changes during the eruption of the tooth, proliferation occurring in its deepest layers, and processes project downwards which become eventually connected with those of the tooth-band and the external epithelium of the enamel organ.

In the description of the microscopical appearance of the tumour it is seen that the structures composing the bulk of the cyst are regarded as epithelial in origin. The majority of the cells of the papillary overgrowths are small in size, rounded, oval, or flattened into a fusiform or

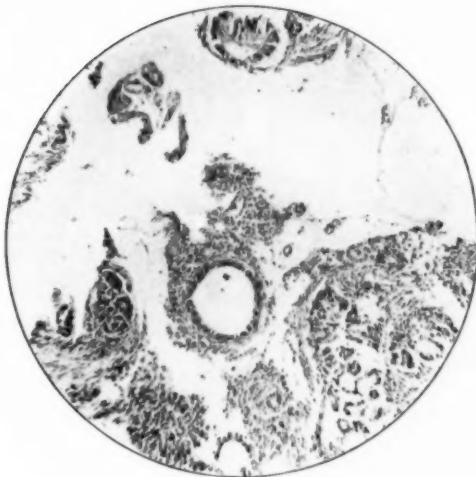


FIG. 6.

Section showing an alveolar arrangement.

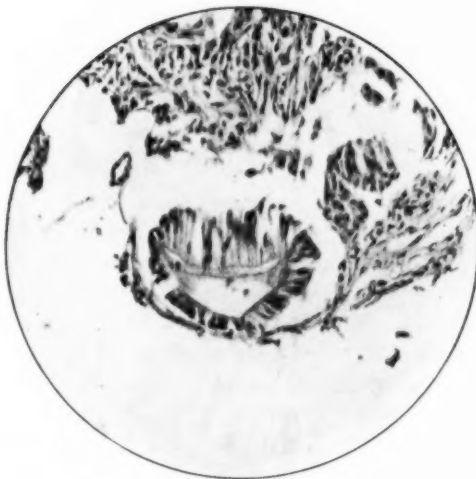


FIG. 7.

Higher magnification of part of the section shown in fig. 6. The columnar character of some of the cells is well seen.

elongated condition. These correspond closely with the cells of the external epithelium of the enamel organ. Many of the cells also appear to be undergoing degeneration, and some strongly resemble those composing the stellate reticulum (fig. 8). The columnar cells lining many of the alveoli of the tumour are probably derived from the internal epithelium of the enamel organ. It is necessary, however, to bear in mind that the tooth-band, which normally extends as a line of cells between the enamel organ and the surface epithelium, is at first formed of an outer layer of columnar cells; these persist for a considerable time in the neighbourhood of the enamel organ, but the typical polygonal cells of the upper part of the tooth-band are not present in the tumour, nor are cells to be found undergoing the changes which normally occur in those of the tooth-band.

We consider that this particular tumour arose mainly in the enamel organ and was associated with changes in the surface epithelium. It seems probable that an extensive proliferation of the cells of the enamel organ has taken place to form a papilliferous cyst. By epithelial overgrowth of the two layers there has developed an abundance of papillary processes and tufts, some of which have become broken off, to form free masses of epithelial cells and alveolar structures. In addition to these changes in the enamel organ proliferations of the surface epithelium have taken place, giving rise to extensive downgrowths in the direction of, and coming into contact with, the papilliferous cyst. The contrast between the two was well shown in sections of the periphery of the tumour, and seems to exclude the surface epithelium from taking part in the origin of the cyst.

When considering the literature dealing with the tumours of the jaw, which are described under epithelial odontome, multilocular cystic epithelial tumour, and other applied names, it is important to determine whether, under their different titles, they possess a common origin, or whether, for purposes of classification, their nomenclature should be distinguished to indicate their different origins. Among the earliest cases described, Falkson [3] concluded that the tumour he examined arose from an enamel organ, or part of it, as the tissues closely resembled it in structure, and that it was not necessary for a whole enamel organ to be involved. Magitot [5], on the other hand, considered that multilocular cysts arose from numerous tooth-follicles. In 1883 Eve [2], giving them the name of multilocular cystic epithelial tumours, stated that, after repeated examination, he had been able to observe in several specimens appearances which distinctly indicated that

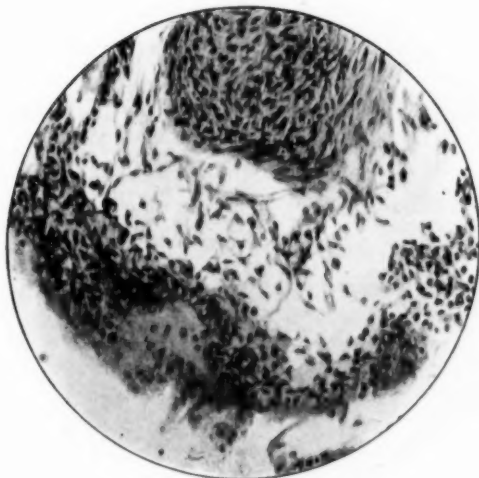


FIG. 8.

Section showing cells resembling those of the stellate reticulum or pulp of the enamel organ.

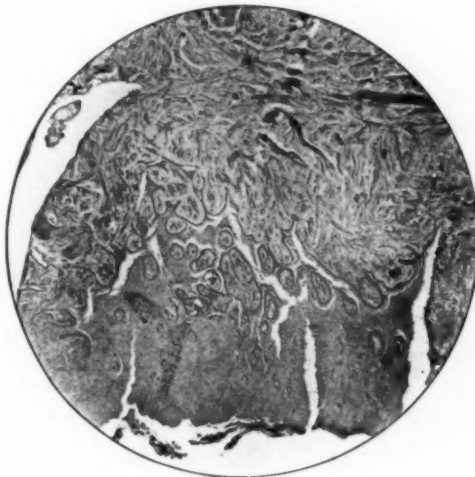


FIG. 9.

Section showing the relationship of the tumour to the gingival mucous membrane.

they originated from an ingrowth of the epithelium of the gum. In 1894 Heath^[4] quoted Malassez's view that multilocular cystic tumours of the jaw had a mode of origin similar to that of dental and dentigerous cysts, and that they arose from an overgrowth of the rudimentary paradental epithelium. Bland-Sutton [1], more recently, came to the conclusion that the majority of the specimens described as multilocular cystic tumours were really endotheliomata, especially in the cases occurring past middle life, and that some of them arose in the gums.



FIG. 10.

Skiagram taken about six months after the removal of the tumour.

The majority of the cases of the tumours collected by Heath are considered by Bland-Sutton to have originated in connexion with the mucous membrane of the jaws. He supported this view by the fact that a certain number displayed malignancy.

In reference to the tumour we are recording, a further statement of Bland-Sutton's is of value—namely, that if these tumours arose in epithelial vestiges of the enamel organ they ought to be met with in the young. This has a direct bearing on our case, the patient being a child

aged 4½. Her future history will be of great interest; deformity or absence of the permanent teeth may occur in the part of the jaw occupied by the tumour. A skiagram, recently taken, suggests the absence of an incisor.

In conclusion, we restate our view that the growth probably had its origin in the enamel organ, and therefore the name "epithelial odontome" should be applied to it.

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- [1] BLAND-SUTTON. "Tumours, Innocent and Malignant" (1906), p. 228.
- [2] EVE. *Brit. Med. Journ.*, 1883, i, pp. 1, 91, 241, 298.
- [3] FALKSON. *Arch. f. path. Anat. und Phys. Virchow*, 1879, lxxvi, p. 504.
- [4] HEATH. "Injuries and Diseases of the Jaws," 1894.
- [5] MAGITOT. "Mémoires sur les kystes de Machoires," Par., 1879.

A Note on the Ameloblast Cells in Esox.

By JAMES T. CARTER, L.D.S.

MR. J. T. CARTER exhibited lantern slides of preparations of the enamel organ in the pike, demonstrating for the first time the existence of processes connecting the lateral surfaces of the ameloblasts.

President's Valedictory Address.

THE PRESIDENT (Mr. Leonard Matheson) said that, before actually concluding the meeting, he had to say farewell to the Fellows so far as the office was concerned that he had, by their suffrages, held for the past few months. In doing so he did not propose to give a formal address, because, at all events in certain quarters, inaugural and valedictory addresses were not looked upon as either useful or desirable; and whatever might be the feelings of those present, he felt that it was not a proper thing to inflict any lengthy address upon them in view of the fact that only four months ago he addressed them inaugurally. There were one or two remarks, however, he might be allowed to make; first

of all with reference to an event which had taken place during the past few months which would stand out as an important event in the history of the Section—namely, the transference of the old museum of the Odontological Society to the hands of the Royal College of Surgeons, which had accepted it as a trust to be held for the Royal Society of Medicine. The Council that evening had received the final communication from the College accepting the trust, practically on the conditions on which the members of the Section offered it to them, through the Royal Society of Medicine, and it only remained for the legal documents to be drawn up for the actual transference to be consummated. He did not think the Section would ever regret that transference, because he could not but believe that, housed and cared for by the Royal College of Surgeons, the museum would be more valuable and of more service to science generally than ever it had been before. He hoped the event would prove that that would be so. He wished to make one reference to the relation of the Section to the Royal Society of Medicine. As President it had been his duty to sit on the Council of the Society, and in attending the meetings of the Council he had been struck with one or two things: first of all by the absolute equality with which all the Sections were treated, the careful consideration that was given to matters brought before the Council by the various Sections, whatever the Sections might be, and the very earnest way in which the Council sought to fulfil its duties in carrying out the high functions and the great responsibilities which the Society had undertaken. With regard to the Odontological Section itself, his short period of office had impressed upon him more deeply than ever the fact that the Section occupied a very important place in the search after knowledge for the sake of knowledge, and in the search after knowledge for the sake of better service to humanity. Those were things worth striving for. They were not only high ideals, but high traditions handed down to them from those who had gone before, and he was glad to think that during next year, at all events, those high ideals and high traditions were sure to be carried on under the presidency of one who had always shown himself devoted to the very best interests of the profession.

PROCEEDINGS
OF THE
ROYAL SOCIETY OF MEDICINE

VOLUME THE SECOND

COMPRISING THE REPORT OF THE PROCEEDINGS FOR THE
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OTOLOGICAL SECTION



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OTOLOGICAL SECTION.

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The Council think it right to state that the Society does not hold itself in any way responsible for the statements made or the views put forward in the various papers.

Otological Section.

December 5, 1908.

Dr. PETER MCBRIDE, President of the Section, in the Chair.

A Case of Thrombosis of the Right Lateral Sinus in which the Clot extended beyond the Torcular Herophili into the Lateral Sinus of the opposite side ; Operation ; Recovery.

By A. L. WHITEHEAD, B.S.

M. L., AGED 38, was admitted under my care to the Aural Department of the General Infirmary at Leeds on September 18, 1907. History : Right otorrhœa for five years, but otherwise good health. Seven days ago felt ill and vomited. General malaise, headache, and occasional vomiting, with elevation of temperature between 100° F. and 101° F.; persisted up to the date of admission. There were no rigors, no convulsions, no delirium.

Condition on admission : A thin, delicate woman, obviously extremely ill ; temperature 101° F., pulse 108, respirations 32. Chest and abdomen healthy. Some tenderness on firm pressure over the posterior border of the right mastoid, and offensive pus in the meatus, with a perforation in the upper and posterior portion of the membrana tympani occupied by small granulations. Optic discs congested, with slight blurring of the edges. No other abnormal physical signs. The radical mastoid operation was performed ; pus, granulation tissue, and carious bone were present in the antrum and mastoid cells, and the caries extended backwards, the dura mater over the lateral sinus and cerebellum being exposed and covered with granulations. The patient's condition being very bad and no rigors having occurred, nothing further was done. The pus was reported to be sterile.

During the next two days the temperature fluctuated between 97.6° F. and 102.2° F. On September 21 the sinus was freely exposed and found to be thrombosed; the overlying bone was removed back to and over the torcular Herophili and the sinus laid open the whole distance; the openings of the longitudinal sinus and of the lateral sinus of the other side were blocked with firm clot, and a small curette passed into the opposite sinus failed to set up bleeding. The internal jugular was exposed in the neck and found to be collapsed; it was tied, a portion being excised and found quite healthy.

After the operation the woman's condition was extremely critical: she appeared almost moribund, and for several days life was only maintained by nutrient and saline enemata, strychnine, &c. During the three days following the operation, a progressive œdema of the opposite side of the face set in, with puffiness of the eyelids, and the superficial veins became gradually dilated, the temporal, facial, and external jugular appearing about the size of a healthy internal jugular. There was intense headache, with a sensation of extreme tension inside the head. The eyes on both sides were remarkably prominent, with retraction of the eyelids, but the conjunctivæ were not congested. There was no definite optic neuritis, but the retinal veins were much dilated. The posterior portion of the wound was quite healthy, but pus continued to discharge from the anterior portion, and there were rigors on September 22, 23, and 24. On September 30, although the rigors had ceased, the temperature continuing to show wide fluctuations and the general condition being very unsatisfactory, the jugular bulb was completely exposed and some purulent clot removed. After this there was slow but progressive improvement, and the wound gradually and completely healed, a trace of pus continuing to be discharged from the tympanic cavity. The patient's general condition, however, remained very unsatisfactory; there was still œdema of the face and scalp, with persistent intense headache and marked dizziness, but the superficial veins were not so conspicuously dilated as during the first two or three weeks.

Early in February of this year—that is, nearly five months after the first operation—fluctuation could be felt under the scalp, and, two long incisions being made, a quantity of pus was evacuated. Between the scalp and the bone there was a mass of breaking down granulation tissue, which was thoroughly scraped away. The abscess was under the periosteum, the surface of the bone being rough, pitted, and exposed from the orbits to the occiput, and laterally to the upper border of the mastoid processes. There was a considerable amount of discharge for

some weeks through these openings, but the scalp gradually became adherent to the skull, and complete healing resulted without any necrosis.

The subsequent history was uneventful. The dilatation of the superficial veins has almost completely disappeared, there is no œdema of the scalp, the headaches and dizziness have passed off, the middle ear is quite dry, and the mastoid wound soundly healed, and the patient in perfectly good health.

DISCUSSION.

The PRESIDENT (Dr. McBride) congratulated Mr. Whitehead on the happy result in the face of such severe lesions.

Mr. A. CHEATLE asked what pathway Mr. Whitehead took in order to expose the jugular bulb. Did he track the sinus down and remove the mastoid process on the way? In some cases it was very difficult to expose the jugular bulb.

Mr. C. E. WEST joined in the congratulations on the result, and remarked that the patient must have had infective thrombosis of both lateral sinuses and the superior longitudinal sinus, and probably non-infective blockage of the cavernous sinus, with diffuse osteomyelitis of the skull-bones. Two cases which he had had threw some light on the œdema of the face. One was a boy with lateral sinus thrombosis on the left side. The sinus was opened up and the jugular tied on that side. He did not get beyond the limits of the clot, though he hoped he was beyond the posterior limit of the infection. The temperature remained very high, the patient was very ill, and Mr. West tied the opposite jugular. The patient died from purulent meningitis due to extension of infection into the cortical veins. There was no apparent result on the intracranial circulation from blockage of both jugulars. Therefore he thought the puffiness of the face must be accounted for by blockage in the orbital veins and the cavernous sinus. He had had one case of chronic osteomyelitis of the skull, with chronic cellulitis of the scalp. Cold abscesses continued to form for eleven months; fourteen incisions were made in the scalp, and post-mortem the vault was so soft that it could be scraped away and the dura mater exposed by a Volkmann's spoon. He died of a frontal abscess, which was not discovered during life.

Mr. WHITEHEAD, in reply, said the result must be partly ascribed to good fortune. He reached the jugular bulb by exposing the lower portion of the mastoid. It was very difficult, but he got it out and cleared right through. He thought the condition of the face must have been due to a non-infective thrombus of the cavernous sinus. But the puzzle of the case was to find out the extraordinary course the venous circulation was going through, seeing that the longitudinal and both lateral sinuses must have been blocked.

Section of a Thrombosed Internal Jugular Vein, which could be felt in the Neck as a distinct Cord-like Structure.

By A. L. WHITEHEAD, B.S.

THE vein has been split longitudinally; a transverse section shows a clot occupying the lumen, consisting of fibrin, with degenerated leucocytes. The adjacent vessel-wall is densely infiltrated with round cells, and the endothelial lining is completely absorbed. The middle coat shows round-celled infiltration. The external coat is normal. There was no periphlebitis.

Mr. WHITEHEAD, in answer to Mr. Cheatle, said that he supposed that the thrombus below where the section was made was a non-infective thrombus, although diplococci were present in it. Still, there was no breaking down.

A Case of Cerebellar Abscess secondary to Infective Labyrinthitis associated with Acute Inflammatory Œdema of the Brain; Recovery; Details of Operative Procedures.

By SYDNEY SCOTT, M.S.

J. C., a male aged 28, was admitted to St. Bartholomew's Hospital on July 15, 1908, with discharge from the left ear and severe headache. History: The patient said the left ear had discharged intermittently since boyhood, and that he had been deaf in this ear for over three years. Nine months ago he began to have severe attacks of vertigo, followed by vomiting and headache. He was unable to walk straight, and had to give up his employment for fear of falling. These attacks were frequent for five months, and for a time they were so severe that he had to keep in bed for two months during last winter. The attacks gradually ceased and the giddiness passed off, so that he was able to resume work in the spring, and has remained comparatively well until a month ago. He then began to suffer from headache. Pain all over the head increased, and he vomited once or twice during the first week. The pain continued more or less, and two days ago

became more severe, especially in the occipital region and in the back of the neck. Yesterday he was sick once. He had had no subjective sensations of giddiness, but he could not walk straight, and was conscious of walking as if intoxicated. He had not had a rigor and had not felt feverish, but he had suffered from want of sleep and loss of appetite.

Condition on admission: The patient walked into the out-patient room with his head in a retracted position. He appeared to be in severe pain. Left ear: Thin, blood-stained pus and polypoid granulations were found in the fundus of the left meatus. There was no mastoid swelling or tenderness, and no abnormally sensitive areas could be detected anywhere on the head or neck. Hearing by bone conduction in the left ear was completely lost. Tuning-fork sounds were conducted to the opposite ear, which was found to be perfectly normal. Rombergism was well marked; the patient fell to the right when standing with feet together and the eyes closed. Gait: When attempting to walk along a straight line with eyes open, he swerved to the right and nearly fell, but was able to recover the erect position. Nystagmus: Spontaneous nystagmus was well marked on deviation of the eyes to the left when the head was erect. The rhythmic movement was concomitant and possessed considerable amplitude, so that the nystagmus elicited when the eyes were directed to the left was quite obvious. The period of movement of the eyeballs alternated regularly, so that each rapid jerk towards the side of deviation was succeeded by a slower movement in the opposite direction. On deviation of the visual axes to the right, with the head erect, no nystagmic movement was noticed at first, but on closer inspection a very fine rhythmic nystagmus was observed; the amplitude of movement was much less than that seen when the eyes were deviated towards the left; the direction of movement on deviation to the right appeared to be purely horizontal, with a distinctly alternating period, the rapid jerk being towards the side of deviation. In a day or two the nystagmus, on deviation to the right, became more easily recognized, and the direction of movement became distinctly oblique and slightly rotatory, with the rapid jerk towards the side of deviation. The fundus oculi: There was no swelling of either optic disc; the inner margins were equally and slightly blurred, an appearance attributable to refraction. The rotation chair and Bárány's tests were not employed as aids to diagnosis in this case. The knee-jerks were equally well marked. There was no ankle-clonus. The plantar reflex was flexor in type. Kernig's sign was considered to be demonstrated,

for the knee-joint could be only slightly extended beyond the right angle when the thigh was semiflexed on the pelvis, the patient being in the reclining posture. After being put to bed the patient became very restless, rolling about and holding his head in his hands. He yawned frequently. The temperature was 98° F. and the pulse-rate 72. Lumbar puncture was performed, but there was no excess of cerebrospinal fluid. A culture tube which was inoculated proved to be sterile after forty-eight hours' incubation. Examination of the blood cytologically showed a leucocytosis of 19,400. The urine did not contain albumin or sugar. A diagnosis was made of infective disease of the left labyrinth, complicated with a cerebellar abscess. Nothing else could explain the patient's condition.

An *immediate operation* was performed on the left mastoid. The antrum was found to contain granulations which led through a wide fistula into a cavity which replaced the canalicular part of the labyrinth. The facial nerve was found to be exposed on all sides and surrounded by granulations (albeit there was no paralysis). Below the nerve granulations were found replacing the cochlea and filling the vestibule. It was necessary to enlarge the fistula behind the facial nerve, to fully expose the unusual cavity which had formed in this part of the petrous, to remove the granulations it contained, and to ascertain that there was no sequestrum. The facial nerve was preserved from injury throughout the operation. No cerebrospinal fluid escaped. The operation on the labyrinth having been completed, attention was directed to the posterior cranial fossa and cerebellum. The dura mater was exposed on the mesial aspect of the lateral sinus by enlarging the bony cavity directly backwards from the region of the labyrinth. The skull was unusually thick and the bone extremely dense wherever it had to be removed. Granulations were found covering the dura mater over an area about the size of a threepenny-piece (1.5 cm. in diameter); around this the dura mater was normal. The softened area of exposed dura mater was penetrated and about 6 c.cm. of very foul pus escaped in a continuous stream. The abscess cavity was within the cerebellum, to which the meninges were adherent. The cavity readily admitted the last phalanx of the forefinger, and was found to have soft, yielding walls. A wide drainage-tube, 1.5 cm. in diameter, was introduced through the opening in the dura mater, but was not projected into the cerebellum any appreciable depth. The wound was packed lightly with gauze and left unsutured.

Subsequent Course.—The patient's headache was greatly relieved by the operation, but the pain returned within two days, and was very severe in the occipital and frontal regions. The patient became very restless and noisy, and required a male attendant. He vomited once. He exhibited well-marked inco-ordinate movements of the left upper extremity. The pulse-rate became slower, falling to 52, and the temperature remained subnormal, 97° F. A *second operation* was carried out to ascertain and remove the cause of these untoward signs. The original cavity was enlarged and the dura mater of the middle cranial fossa exposed, and the dura mater of the posterior fossa and the sigmoid sinus widely exposed. The cerebellar abscess cavity was explored with the last phalanx of the forefinger; its walls appeared to have encroached on the cavity, and were everywhere soft and almost diffuent. No more pus escaped. The temporo-sphenoidal lobe was punctured in the direction of the lateral ventricle, which was not found to be distended. One felt justified in attributing the symptoms of compression to acute inflammatory œdema of the brain; having regard to the almost invariably fatal termination of this complication, it was decided to enlarge the opening in the dura mater to permit herniation of the underlying cerebellum, and so afford some relief to the intracranial tension. The dura mater was incised horizontally from the internal auditory meatus to the descending limb of the lateral sinus. No tube was inserted, but the cavity was lightly packed with gauze. The earlier part of the operation was hampered by profuse hæmorrhage from the lateral sinus, which was accidentally nipped with bone forceps before the vessel was fully exposed.

Subsequent Course.—The following day the patient complained less of pain in the head, although he still had considerable suboccipital pain and stiffness of muscles at the back of the neck, and the retraction of the head was still present. He had several attacks of hiccuping. Eventually he was able to sleep, which he had not been able to do for several days. A large hernia cerebelli rapidly formed and nearly filled the operation cavity; at first there was no pulsation, but as the headache diminished the pulsation of the hernia and of the dura mater became evident. Weakness of the left side of the face gradually developed, and eventually passed into complete paralysis. Kernig's sign and the retraction of the head disappeared with the headache and stiffness in about a week, when the hernia also ceased to increase in size. The leucocytosis persisted until the second week: the count was 18,000 on the ninth day. The actual exudation of pus was always small in

quantity after the abscess was first opened. On the thirteenth day the leucocyte count was 7,800. The nystagmus diminished in intensity, but possessed the same characters as those noted on admission for two or three weeks. In the course of time the nystagmic movements elicited by visual deviation to the right became more evident, while those resulting from deviation of the eyes to the left became less marked. At the end of the second fortnight the post-aural wound was entirely closed with secondary suture; the cavity was packed through the enlarged meatus. The wound and cavity rapidly healed, and ten days later the patient left the hospital. He could walk quite straight and had regained co-ordinate control. His reflexes were normal, and there was no hypotonus or other evidence of cerebellar deficiency. The patient now feels perfectly well and has returned to his work. The facial paralysis is the only defect which concerns him; though it is still possible that some power may return, no signs of improvement are yet manifest.

DISCUSSION.

The PRESIDENT said the operator was to be congratulated on the successful termination of the case. It was difficult to have an adequate discussion on a case which lent itself only to praise.

Dr. W. MILLIGAN asked whether Mr. Scott proposed to do anything with the hernia of the cerebellum, which was projecting into the auditory meatus. He agreed that the result was admirable. A few years ago he had a very similar case in a young woman between 20 and 30 years of age. The abscess was approached from behind, through the posterior wall of the petrous, and a large hernia of the cerebellum resulted. He first tried cutting away more bone and making a large hole in the occipital region, but the hernia continued. Finally, he applied a lead plate and graduated pressure, with the result that the hernia disappeared and the patient made a good recovery.

Mr. A. L. WHITEHEAD said he thought the case showed the importance of approaching a cerebellar abscess through the posterior wall of the bony cavity rather than making a separate opening behind the lateral sinus. Here the abscess was in close contact with that portion of bone. It was stated that at a subsequent operation the cerebellum was explored with the finger. It was difficult to know what to do in the face of the œdematous condition of the brain; there might be loculi of pus, and by putting the finger in, more tissue might be broken down and the spreading œdema increased thereby. But this patient's vitality seemed to have been great enough to overcome that spreading œdema. Such œdema was often the cause of death in such cases. He thought it must be owing to defective drainage.

Dr. DAN MCKENZIE desired to direct attention to the interesting problem presented by the nystagmus. It had been enunciated as a law that destruction of the labyrinth on one side induced spontaneous nystagmus to the other (the sound) side, and that nystagmus due to cerebellar abscess was directed towards the same (the diseased) side. In the case under discussion both lesions had been present, and yet, while there had been well-marked nystagmus to the diseased side, the nystagmus to the sound side had been but slightly marked when the patient first came under Mr. Scott's care. In other words, while the cerebellar nystagmus was pronounced, the labyrinthine nystagmus was trifling, although the destruction of the labyrinth was, comparatively, as extensive as that of the cerebellum. Did the effects of the cerebellar lesion on the nerve-centres governing nystagmus counteract those of the labyrinthine lesion? The question could not be answered in the affirmative from the facts before them, for it was possible to explain the feebleness of the labyrinthine nystagmus in another and more probable manner. The history of the case informed them that several months before the date when the first examination was made the patient had been attacked with vertigo so severe that he was compelled to take to his bed. Evidently this attack had been caused by the onset of acute labyrinthitis, and, doubtless, if an examination of the eyes had been made at that time violent labyrinthine nystagmus would have been discovered. Some time later this nystagmus probably became less marked as the case passed into what Bárány had called "the stage of latent labyrinthine destruction." At the present moment there was still some slight rotatory nystagmus on extreme lateral deviation of the eyes towards either side, a circumstance which might be looked upon either as due to the still lingering traces of the violent nerve-storm which the grave and disturbing lesions had evoked, or simply as a normal condition, seeing that 60 per cent. of all individuals manifest slight nystagmus on extreme lateral deviation. It was, perhaps, unfortunate that the caloric and rotation tests had been omitted, not because they would have influenced the decision in this particular case, but because a double lesion of labyrinth and cerebellum was uncommon, and the results of the caloric and rotation tests might have supplied a help in the diagnosis of similar lesions in the future in cases where the other clinical features were less undoubted than they had been in this case.

Dr. KELSON remarked on the facial paralysis, which curiously only came on just as the other symptoms were subsiding, and he asked why that was. Did Mr. Scott consider it was connected with the disease in the middle ear or with the incision which he made horizontally from the internal auditory meatus to the lateral sinus?

The PRESIDENT asked Mr. Scott whether he considered that the hard and fast rules which had been laid down by, he thought, Neumann as to the direction of nystagmus in the diagnosis between cerebellar and labyrinthine lesions were so established as to be of absolute use in the diagnosis of difficult cases.

Mr. SCOTT, in reply to the President, said that the type of nystagmus in this case of destructive disease of the labyrinth associated with cerebellar abscess was different from that which he had met with in cases of destruction of the labyrinth alone. On several previous occasions the President had drawn the attention of members to observations of Continental observers and others that cerebellar disease on one side had been associated with nystagmus elicited by deviation of the eyes to the *same* side. That had been so in this case. It was because he observed this, in association with other signs, when he first saw the patient that he suggested the probable presence of a cerebellar abscess complicating infective labyrinthitis. In reply to Dr. Milligan, he did not propose to do anything to the small cerebellar hernia, which had completely healed over, was not getting larger, and was confined to the external auditory meatus, which it practically obliterated. Regarding the point raised by Mr. Whitehead, at the second operation he (Mr. Scott) attributed the symptoms of increased intracranial pressure to acute inflammatory oedema of the brain. Another possibility was ventricular distension, which he excluded by puncture of the lateral ventricle. There might have been a second abscess, and if encapsuled it might have offered resistance; it was for this reason he explored the cerebellum with the finger and found it unusually soft, almost diffuent, and there was no sign of another abscess. He therefore purposely enlarged the opening in the dura mater to allow the cerebellum to herniate freely with the hope of relieving the intracranial tension. This procedure had had the desired effect, and the patient had recovered. He agreed with Dr. McKenzie that, according to the history, nine months before the operation the patient must have had infective labyrinthitis, and that for some time nystagmus would no doubt have been present on deviation of the eyes to the *opposite* side. It would only be after the more recent invasion of the cerebellum that nystagmus on deviation of the eyes toward the *same* side developed. In answer to Dr. Kelson, the facial paralysis was not present before the operation, nor was it present the following day. It only very gradually developed on succeeding days. It was not due to injury: the nerve, which lay bare in its sheath among the granulations, was preserved absolutely intact throughout both operations. He attributed the palsy either to the possible interference with the blood-supply or to inflammation within the sheath of the nerve. It was too soon to speak of the ultimate issue; though he hoped it would recover, he feared there might not again be true symmetry of the face.

A Case of Infective Meningitis, secondary to Infection of the Labyrinth, successfully treated by Translabyrinthine and Lumbar Drainage.

By C. ERNEST WEST, F.R.C.S., and SYDNEY SCOTT, M.S.

A LITTLE girl, aged 7, was admitted to St. Bartholomew's Hospital on February 6, 1908, with long-standing purulent discharge from the left ear, to undergo treatment by the radical mastoid operation. She had presented no recent symptoms suggestive of labyrinthine disease. At the operation granulations were found filling the pelvis ovalis, destroying the stapes and eroding the Fallopian aqueduct. The open fenestra ovalis was enlarged by the removal of bone inferiorly without interfering with the cochlea or semicircular canals. When the inner wall of the vestibule was curetted, cerebrospinal fluid escaped freely. The patient was unusually sick after the operation, and continued to vomit slightly for five days. Cerebrospinal fluid escaped into the dressings for six days. The post-aural incision healed by primary union. Facial paralysis appeared on the third day. A week after the operation the child complained of frontal headache, which became constant and severe, and retraction of the head developed. Kernig's sign was well marked, the knee-jerks were suppressed, and the plantar reflex was extensor in type. The temperature rose to 103° F., and the pulse-rate to 112.

Second Operation (February 18): Completion of "double vestibulotomy" and translabyrinthine drainage. The original operation cavity was reopened, the external and superior semicircular canals were cut away and the vestibule opened above the facial nerve. The fundus of the internal auditory meatus was opened by removal of the antero-internal wall of the vestibule. A large quantity of cerebrospinal fluid escaped under pressure from the opened internal auditory meatus. A wire, doubled and spirally twisted, was introduced as a drain into the internal auditory meatus through the open vestibule. The immediate effect was remarkable. Headache and retraction disappeared. The temperature sank to 98° F., and the pulse to 88. Vomiting ceased. Two days later the triad of symptoms—headache, retraction, and Kernig's sign—reappeared. The temperature rose to 101.8° F., and the pulse-rate

to 92. There was no squint or nystagmus; the pupils were equal and active; knee-jerks were absent. During the next two or three days these symptoms were associated with the intermittent retention of cerebrospinal fluid; after each dressing and manipulation of the wire drain there was a free escape of cerebrospinal fluid into the dressing, with temporary disappearance of headache and retraction. On February 25, a week after drainage through the internal meatus had been established, the triad of symptoms became more marked, the child crying out continually with pain in the head. Lumbar puncture was performed, and turbid fluid was withdrawn. Cytological count showed an excess of polymorphonuclear leucocytes (62 per cent.) over lymphocytes (27 per cent.). No organisms could be recognized in films of the centrifugized (on February 25 and on February 27) fluid or in cultures. There was a high blood-leucocytosis of 45,000. There was no optic neuritis. After the withdrawal of fluid by lumbar puncture for examination the child was relieved, as she had previously been, by the flow of fluid from the basal meninges.

Third Operation: On February 26 the possibility of a cerebellar abscess was excluded by exploration. Continuous drainage of the lumbar theca was established by a fine cannula introduced between the laminae of the fourth and fifth lumbar vertebrae, the outer end of the cannula being enveloped in a Keith's dressing. Subsequently the translabyrinthine and lumbar drainage wounds were dressed daily. February 27: The large pads of gauze and wool within the Keith's sheet were wrung out into a measuring glass; about $\frac{1}{2}$ pint of turbid fluid was expressed from them. General condition much improved. February 28: The quantity of fluid escaping from the lumbar theca continued to be very large, but it was now clear. Temperature 97° F.; pulse 80. Child taking fluid very freely. There was now no headache, and the head could be freely flexed on the chest. The child remained quite comfortable, but became very weak. On the night of February 29 there was severe collapse: the child was cold and listless, pulse irregular and hard to count; vomiting reappeared. There was no headache, and the child was quite rational when roused, but intensely weak, and apparently dying. The cannula was removed from the spinal canal after eighty hours' continuous drainage. The pelvis was raised and head lowered; hot saline and brandy were given by the rectum and hot milk and water by the mouth; strychnine hypodermically. During the next twelve hours 3 pints of fluid were taken by the mouth, as well as a pint of saline *per rectum*.

The child rallied marvellously, and on March 3 the wire drain was removed after fourteen days' drainage of the basal meninges. The subsequent course was uneventful, except for a minor plastic operation to close the post-aural wound, which had been left open after the second operation. She was discharged on April 8. The facial paralysis has much improved. The ear is dry, and in general health the child is perfectly well.

DISCUSSION.

The PRESIDENT said the case raised some points of considerable interest, and he asked whether there were any marked labyrinthine symptoms at the beginning of the case. Was it shown that the foot-plate of the stapes was destroyed at that time? Most members would remember the long paper by Zeroni, from Schwartz's clinique, on post-operative meningitis, and in it, it was shown that in cases of radical mastoid operation it was especially dangerous to scrape granulations on the inner wall of the tympanum. He asked the authors' opinions as to the course the case would have pursued had the inner wall of the tympanum and the labyrinth been left alone at the first operation.

Mr. A. CHEATLE said the case was most interesting. With regard to infection of the labyrinth, he thought it was due to surgical enthusiasm. No record was given of tuning-fork tests before the operation. Every case of suppuration before being operated upon should be tested in that way. No doubt it was an infective meningitis. Possibly it was a good thing that meningitis did occur, because of the treatment applied, which, as far as he knew, was quite new. In future, he would adopt the same procedure in similar cases of his own.

Dr. KERR LOVE said he thought the authors were to be congratulated on the fact of recovery, because of the light it threw on the treatment of such very difficult cases. It was usual, he thought, to look upon meningitis as too serious a condition. With middle ear suppuration meningitis was extremely serious, but, even without operation, it was not necessarily fatal. A year ago he had a case of meningitis in which the diagnosis was verified by lumbar puncture, and which recovered without operation. Meningitis without middle ear suppuration was not so serious or fatal as it was usually considered, otherwise meningitis would not occur without middle ear suppuration as the most common cause of deafness or deaf-mutism in every country. The meningitis which caused deafness and did not kill was not tuberculous, and he thought more information was wanted about that variety. Was it a disease which had long been recognized in America and Germany as cerebrospinal fever? He, like Mr. Cheatle, would now open the canal and leave the condition to drain, instead of performing lumbar puncture repeatedly.

Mr. WAGGETT said he had employed continuous lumbar drainage—on one occasion for five days, about a year ago—with immediate excellent results; but

he thought there should be caution as to the amount of fluid drained from the lumbar region. His patient became very much exhausted on two or three occasions, and he was relieved by temporary corking of the tube. The patient died with empyema of the right lateral ventricle, infected from the corresponding cavity on the left side, which cleared up under free drainage; nevertheless, the speaker was convinced, by the repeated improvement of symptoms, that continuous lumbar drainage was a valuable procedure and one which presented no serious practical difficulties.

Mr. A. L. WHITEHEAD asked whether any examination was made of the cerebrospinal fluid which escaped at the second operation. Also, was the fluid which escaped after the first operation clear? Possibly it did not become purulent until about a week later.

Dr. W. MILLIGAN said there was a note that the temperature was 109° F. and the pulse-rate 112, which gave the impression that the condition must have been fairly localized.

Mr. WEST, in reply, said the case was not brought as a triumph, but as a suggestion, and, indeed, an experiment; 109° F. was a misprint for the temperature—his recollection was that it was 103° F. He hoped the proceeding might be useful in mild meningitis and in cases of cerebrospinal meningitis, from which so many died, or, if they recovered, were crippled by being deaf. There were no initial labyrinthine symptoms, and the foot-plate of the stapes was certainly not in situ. Possibly the case would have done better if the inner wall of the tympanum had not been interfered with, but he and his colleague had had several lamentable cases, in which the inner wall of the tympanum had received no particular attention, which developed acute labyrinthitis and meningitis, dying of the latter. The tuning-fork reactions were not stated, and he admitted that ideally every patient should have a complete register of such reactions carried out; but the child was only 7 years of age, and on one so young the results were generally worse than useless. Another case which recovered from meningitis under similar treatment died of pneumonia later. The patient in question to-day was nearly killed by the drainage of the fluid, and since then their practice was to compensate for that by giving large quantities of fluid by mouth and rectum. The case began as a serous meningitis and then became sero-purulent, the fluid becoming more and more cloudy.

Mr. SCOTT, in reply, recalled the case of a boy who died nearly two years ago with acute internal hydrocephalus, and which he had published fully (*Archives of Otolaryngology*, 1908, xxxvii, p. 108). This case first directed their attention to the possibilities of drainage of the basal meninges. Had the labyrinth been opened, and the sheath of the arachnoid around the seventh and eighth nerves and basal arachnoid cisterns been drained, he believed that patient also would have recovered. He had seen other similar cases. He agreed that otitic meningitis was viewed too hopelessly, and he was quite sure that there were certain cases which were recoverable. He was accustomed to regard

two chief forms of otitic meningitis: the one in which the outstanding features were masked by septicæmia or toxæmia, and the other which was characterized by symptoms of increased intracranial pressure due to ventricular distension. It was the latter class which they hoped to be able to relieve by removing the primary focus of infection and by drainage of the basal meninges, &c. In many cases of cerebrospinal meningitis and post-basic meningitis, he had found by histological examination of the petrous after death that the channel of infection to the meninges was through one labyrinth. He had reported such cases at the last meeting of the Section, and shown histological specimens with round-celled infiltration in the scalæ of the cochlea and in the modiolus. Dr. Love had spoken of deaf-mutism following mild cases of cerebrospinal meningitis. Mr. Scott had specimens and sections which he believed threw light on the nature of this type of deafness.

A Case of Purulent Encephalitis treated by Drainage and Removal of Infected Brain-tissue.

By C. ERNEST WEST, F.R.C.S.

MALE patient, aged 17, admitted to St. Bartholomew's Hospital September 18, 1908, with bilateral otorrhœa and headache. The otorrhœa had been present for at least two years; headache for five or six weeks, worse during the last five days, diffuse but worse on left side. There had been drowsiness for four days, during which time there had been several rigors. No history of vomiting, vertigo, or tinnitus. Looks recently ill, tongue thickly coated. Temperature 100° F.; pulse 85, good tension, regular, no dicrotism. Respiration very shallow, 24. *Ears*: No swelling of meatal walls; both membranes gone, irregular warty granulations on inner tympanic walls. Slight tenderness of left mastoid on firm pressure, none on right. No mastoid œdema. *Eyes*: No nystagmus; no squint. Discs both slightly congested, but not swollen. *Head and neck*: No paralyses of cranial nerves; no retraction of head. Some enlargement of upper deep cervical glands on both sides without tenderness. Knee-jerks equal, not exaggerated. Grips equal. No inco-ordination. Mental condition: Answers questions slowly, but with clearness, and without difficulty in use of words. Leucocytosis, 21,000.

Operation carried out immediately on left side. During initial radical mastoid operation a large supratympanic abscess was opened. Dura above the tegmen necrotic over an area about 1 cm. in diameter,

fistula through this area leading into the temporo-sphenoidal lobe. Fistula enlarged, and a small amount of very smelly pus evacuated; there was no definite abscess cavity, the surrounding brain being sloughy and ragged. Rubber drainage-tube sutured in position, wound left open and cavity packed from behind. Five hours after the operation the patient could answer questions rapidly and accurately, but could not remember where he lived. Headache relieved.

There were some fluctuations of condition during the next four days. The wound was very offensive, and was treated by fomentations. On the evening of September 22 his temperature rose suddenly from 98° F. to 104.6° F.; there was no rigor. He became very drowsy, but when roused complained of frontal headache. Pupils equal, moderately dilated; no squint; no retraction. Pulse relatively slow, 80, when temperature 104.6° F.

Second Operation: At 10.30 p.m. wound reopened. Widespread but thin layer of pus found between dura and bone; whole of squama removed. Free incision of dura mater and exploration of region of abscess. The interior of the brain was found to be sloughing, and stank horribly. No further collection of pus found. A large window was cut out of the dura mater, which had been in contact with the tegmen and squama, about the area of a half-crown, and the underlying cortex and white matter were freely scooped away with a large Volkmann's spoon until the sloughing area was widely exposed. This resulted in the descent of the exposed surface into the opening and the provision of excellent drainage from an otherwise inaccessible part. The cavity was filled with gauze and the patient returned to bed in a much improved condition. There was no noticeable shock.

Subsequent course: Temperature fell by lysis to normal during the next forty-eight hours; disappearance of headache; daily dressing; the brain did not herniate, but remained at the level of the gap in the dura mater; sloughs of brain and dura mater slowly separated. Secondary suture of the wound on October 14. Sent to convalescent home November 3. Mental condition quite satisfactory; no evidence of any paralyses or disability.

DISCUSSION.

The PRESIDENT said the Section was much indebted for the case. It was a new surgical departure to cut a window out of the dura and remove the breaking down cerebral substance.

Mr. WAGGETT said that a question of great practical importance arose out of the case. He had seen some cases comparable with the present one, and asked why it was that adults usually got well of the condition, while in children a gradually spreading encephalitis nearly always occurred. Was there any method of stopping the gradual spread in children? He had had perhaps half a dozen cases in children who had gradually sunk, and died several days after operation. He could at the moment recall only one case of recovery in a child, and that was one which had been in coma for ten days. The patient lingered on for three weeks, the encephalitis gradually spreading. He then went for his holiday, and on his return found the child just back from the convalescent home and in good health. He was forced to surmise that this unexpected recovery was not unconnected with the cessation of active measures directed to the cleansing of the diseased parts.

Dr. A. BRONNER asked whether there were diplococci in the pus. He believed one theory to account for brain abscesses having such thick capsules was that diplococci were present in large numbers—the more diplococci there were present, the thicker the capsule was.

Mr. HUNTER TOD said everyone agreed that when pus was known to be present it should be let out; therefore, if there was pus beneath the dura mater the proper procedure was to cut away bone and freely open the dura. There was nothing new in this. He doubted whether it was correct to curette the brain substance, though, of course, there should be drainage, as he thought that by doing so there was a risk of infecting the surrounding brain tissue and of thus setting up further œdema. He considered, therefore, that the brain in the first instance should be left alone, but if hernia occurred later on, removal of the projecting portion of brain was justifiable.

Mr. WHITEHEAD agreed with Mr. Tod's remarks. The case was on much the same lines as Mr. Scott's cerebellar case, and Mr. Scott apparently gave free drainage without removal of brain matter. In adopting the latter procedure he thought Mr. West was exposing the patient to unnecessary risk. Spreading œdema was more common in cerebellar conditions than in cerebral, and in children the former was the more common.

Dr. KERR LOVE said a case he had at present under care had some bearing on the present discussion. A child in his ward had the radical mastoid operation done on one side, and during the process of recovery the other side went wrong. While awaiting operation on this side an abscess developed. At the operation this abscess was found to have opened through the dura mater, and a large part of the floor of the middle fossa was removed. The temperature remained up, but the abscess healed, and the temperature was latterly better than since the patient's admission. No brain matter had been removed, and free drainage promised to cure the local condition.

Mr. WEST replied that he had seen three adults die from spreading œdema, with obvious infection and decomposition of the brain substance. The brain substance taken away in the present case grew *Streptococcus pyogenes* in pure

culture. He admitted the whole problem was one of drainage, but if there were stinking sloughs inside the brain he did not think any mere drainage-tube would remedy matters. The case was desperate, and he wanted to bring the sloughs to the surface. The infected surface came down into the window and wept decomposing serum, and the sloughs were able to be cast off. There was no increase of intracranial pressure in the case. The patient now had no headache, and could shake his head about without provoking symptoms. He had not seen any bad results from adhesions in temporo-sphenoidal abscess. In this case the adherent area of brain was not cortex, but white matter.

Specimen and Notes from Case of Tumour of the Auditory Nerve.

By MACLEOD YEARSLEY, F.R.C.S.

A FEMALE patient, aged 46, was sent to me on May 4, 1905. She complained of deafness in the left ear for fifteen months, with tinnitus of a variable character and likened to a sewing machine, combined with clicking and swishing. There was no vertigo, no paracusis Willisii. She stated that she first discovered her deafness by failing to hear her watch, which she was in the habit of often putting to her ear. Both membranes showed slight dullness, but were not indrawn or impaired in mobility. The nose and throat presented nothing of note. There was nothing in the personal or family history of note.

Functional testing gave the following results: Weber, R. positive; Rinne positive to C and C₂ on the R.; negative to C, positive to C₂ on the L. Bone conduction, C (128) R.—7 sec.; L.—26 sec. Edelmann-Galton pfeife, R. 50,000 vibrations; L. 10,000 vibrations. Acoumeter, R. over 10 ft.; L. 0. Voice, R. over 10 ft.; L. 2 in. Whisper, R. 35 in.; L. 0. By air conduction, R. 3C(16), 0; 2C(32) just heard; 1C(64)—20 sec.; C(128)—4 sec.; C¹(256)—7 sec.; C²(512)—5 sec.; C³(1024)—4 sec.; C¹(2048)—14 sec.; L. 3C(16) to C(128), 0; C¹(256)—30 sec.; C²(572)—27 sec.; C³(1024)—20 sec.; C⁴(2048)—25 sec.

The patient was placed on strychnine, and in June, 1905, other nerve symptoms appeared, especially unsteadiness of gait, diminution of knee-jerk and right ankle-clonus. I saw her again on June 28, 1905. Her tinnitus was much diminished, slight whistling only being complained of. Bone conduction for the C(128) fork was R.—15 sec.; L.

—23 sec. I wrote to her doctor suggesting that she should be seen by a neurologist. She did not, however, see Dr. Campbell Thomson until last May (1908), to whom I am indebted for the following notes: About three years ago first noticed a little difficulty in walking, with a tendency to lurch to the left. She saw Sir John Tweedy two years ago, and again in April, 1908, when double optic neuritis was discovered. She has occasional pains over the top of the head, but no severe headache. For the last six months she has had occasional sickness. Complaints of tremors of the left hand on using it. For the past three days has had an unpleasant taste in the mouth. Signs: left deafness. Feeling of numbness over second and third division of the fifth nerve; no anaesthesia; no facial weakness; no double vision, but has noted it; nystagmus marked to left, (?) very slightly to right; double optic neuritis; pupils react to light; tremors on holding out a book; gait reeling, with lurches to left; left knee-jerk slightly more brisk than right.

For the following tests, taken on May 9, 1908, I am indebted to our late house surgeon, Dr. G. N. Biggs. Weber, R. positive. Rinne (C), R. positive, C and C₂; L. negative. Bone conduction C(128), R.—14 sec.; L.—30 sec. Air conduction: R. 3C(16) to 1C(64) diminished; C(128)—35 sec.; C¹(256) to C⁴(2048), diminished. L., 3C(16) to C¹(256), 0; C²(512), perception; C³(1024) and C⁴(2048), very much diminished. Acoumeter, R. 10 ft.; L. 0. Voice, R. 12 ft.; L. 0. Whisper, R. 1 ft.; L. 0.

The tumour shown was removed by Sir Victor Horsley; it was growing from the sheath of the left auditory nerve and occupied the lateral recess. The patient rallied but died on May 30, three days after the operation, from sudden cardiac collapse.

The pathologist's report says: "This tumour is a fibroma, such as is known to arise from the sheaths of nerves. It is comprised of interlacing bundles of fibres and areas of mucoid softened tissue. There is no reason to regard the growth as malignant."

A Syringe for Use after the Radical Post-aural Operation.

By URBAN PRITCHARD, F.R.C.S.

THE opening of the nozzle of this syringe is at the side of the tip, so that the direction of the injection is sideways, not straight forward, thus resembling an intra-tympanic syringe, but the current is larger

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and the force greater. By its use the posterior cul-de-sac of the cavity can be easily washed out.

DISCUSSION.

Dr. DUNDAS GRANT said it was a very valuable instrument, and he was only surprised that such an obvious device should not have been thought of before.

Dr. BRONNER suggested it was dangerous to use too much force in the region concerned.

Mr. CRESSWELL BABER said it was an ingenious instrument, but thought there should be some indication of which side the stream was being projected from.

Dr. URBAN PRITCHARD replied that the obvious answer to Dr. Bronner was that too much force should not be used in anything. He did not think that any harm could be done with the instrument. Mr. Baber's point was met by a mark on the nozzle of the syringe to indicate the position of the opening.

Anatomical Specimens illustrating the Surgical Anatomy of the Temporal Bone.

By ARTHUR CHEATLE, F.R.C.S.

THE collection consisted of 525 temporal bones of all ages, sectioned and dissected to show the variations in the anatomy bearing on the spread and cure of suppuration.

DISCUSSION.

The PRESIDENT expressed the gratitude of the Section to Mr. Cheatle for the exhibition of his interesting and valuable collection.

Dr. GRAY congratulated Mr. Cheatle on his splendid collection, and expressed the wish that Mr. Cheatle would give a demonstration on his series, as the subject was so important.

Mr. CHEATLE replied that he was delighted to show the specimens at any time, and he had brought them now because the series was going to be broken up for additions.

Case of Temporo-sphenoidal Abscess with Maniacal Symptoms, opened up through the Tegmen Tympani, washed out and drained, and treated with Iodoform.

By ADOLPH BRONNER, M.D.

GIRL, aged 11, had discharge from left ear for nearly two years. Last few months pain in ear and head. Two days very excited, and, if touched or spoken to, screams and fights, and throws herself on the floor. Papillitis. Pulse 42. Mastoid opened; small cholesteatoma of attic. Tegmen tympani removed. Dura thickened and slightly adherent. Incision. Pus searcher found pus $\frac{1}{4}$ in. above dura; cavity washed out and iodoform emulsion injected. Drained and iodoform injected for eight days; in four days recurrence of pain; cavity reopened and much clear, bloody serum escaped. Points of interest: (1) Maniacal symptoms when talked to; (2) abscess opened through tegmen tympani; (3) cavity refilled with non-purulent serum after eight days' draining. This speaks in favour of draining and washing out cavity and injecting iodoform.

DISCUSSION.

The PRESIDENT congratulated Dr. Bronner on the result. Maniacal symptoms were, in his experience, very rare in such cases.

Dr. DUNDAS GRANT agreed that maniacal symptoms were uncommon in such cases, but in one case which he showed before the Otological Society, the maniacal symptoms were very striking, illusions rather than delusions being the feature. The lesion in that case also was near the surface, and recovery came about with comparative ease.

Mr. TOD asked what Dr. Bronner's views were on the question of irrigation of a brain abscess, concerning which much difference of opinion existed. Did Dr. Bronner always irrigate, or on what occasions did he do so? Also, what were his comparative results from irrigation and non-irrigation?

Dr. GRAY asked whether there was a family tendency towards mental disorder, as such a case might have been due to that.

Dr. BRONNER replied that he did not generally irrigate, but in this case there was a large cavity and much offensive pus, also the cavity had a thick wall. One should be sure there was a thick wall before irrigating. He hoped to hear the question discussed of gauze versus a drainage-tube. Where there was a thick capsule he would use a tube, but when encephalitis was present

he would use gauze. Twelve days after the operation the child had symptoms of brain trouble (pressure), and when the abscess cavity was reopened bloody serum escaped. He did not think there was any history of mental disability in the family, and the patient was now all right.

Disease of External Auditory Meatus in a Girl.

By W. H. KELSON, M.D.

A. W., a girl aged 10, never had discharge from the ears; no evidence of syphilis, hereditary or acquired. A week ago severe pain felt in right ear and afterwards in left. Both external auditory meatuses became red and swollen and covered with a white deposit difficult to remove; no discharge was present; mouth and pharynx normal.

Otological Section.

February 6, 1909.

Dr. PETER McBRIDE, President of the Section, in the Chair.

Dermoid of Mastoid Region.

By P. McBRIDE, M.D.

Miss L., aged 21, dark hair and eyes, complained of a swelling behind the right ear. On examination the right auricle was seen to stand out to an unusual extent; on inspecting the back of the ear a swelling was noted occupying the angle between the upper part of the auricle and the base of the mastoid; the eye was at once attracted by its bluish colour, and it looked exactly like a very large vein; on palpation it was felt to be about as large as a big hazel nut; it was freely movable and not adherent to the skin, which could be pinched up all over the tumour, and was fluctuating; it felt as if its size could be influenced by pressure, but it could not be dissipated entirely; I may say at once that this must have been an error resulting from its very free mobility. I had little doubt the growth was an angeioma, and therefore removed it very carefully, raising a flap of skin; the bluish colour was then even more marked and reminded me of an exposed lateral sinus; at the angle between the mastoid and auricle a vein of some size entered the growth; this was ligatured; after the stitches had been tied considerable bleeding occurred from another vessel in the upper and anterior part of the wound; the stitches were removed and the bleeding stopped by passing a stitch of silkworm gut under the vessel and making it also connect the corresponding edges of the incision. The growth, which still retained its bluish colour, was placed in formalin; on cutting into it I was surprised to find the fluid contents to consist of a yellowish, syrupy, semi-fluid substance, containing numerous hairs. The mother told me

the growth had been noticed like a small "vein" from early childhood, but that recently it had begun to grow bigger and push forward the ear. There were no symptoms complained of. Microscopic examination showed epithelium and hairs, but the latter were not particularly dark in colour; no pigment was seen, but the cyst-walls were excessively vascular, and to this we must probably attribute the dark colour.

DISCUSSION.

Dr. W. MILLIGAN said he thought dermoids in that situation were very rare; he had only seen one case. Retention cysts were, however, fairly common. An interesting point in connexion with the diagnosis of a dermoid cyst was the effect of transmitted light, which revealed somewhere in the dermoid a little dark mass. If the dermoid was removed and that part examined, it was found to be a concentrically-arranged mass of cells, probably "inclusion" cells connected with the branchial cleft. The skin was freely movable over a dermoid. Some time ago he had a case, which he showed before the Otolological Society, in which the cyst had undergone malignant degeneration. The pathologist who examined the specimen thought it was originally a dermoid cyst, and that it had undergone malignant changes. The patient was a girl of 19, and the condition had been noticed for years.

Dr. E. LAW said it would be interesting to hear if other members had seen a dermoid cyst in this situation; he had not.

Mr. CUMBERBATCH said he had seen many cases of true dermoid cysts, but never one in the mastoid region, and in spite of what had been written as to its frequency he should consider it rare in that region.

Dr. URBAN PRITCHARD said he had never seen a similar case. He thought the reason they were so often described in books was that the descriptions had been copied from book to book, and the authors of those books had probably not seen a case.

Mr. MARTINEAU said that many years ago, when he was house-surgeon at St. Thomas's, he assisted Mr. Makins with a case in private, a case of dermoid cyst over the mastoid. The skin was freely movable, and there was a small indentation in the bone in which the cyst lay. The cyst-wall was adherent at this spot, but otherwise dissected out easily. The wall was thickened by recurrent attacks of inflammation, and the contents were sebaceous matter and fine colourless hairs.

Mr. CHEATLE said he had dissected out a thick-walled cyst from behind the ear which had been there since infancy. It had a white thick wall. He removed the granulations from Shrapnell's membrane, and there was fine hair over it. He believed Dr. Pegler had shown a similar specimen, with fine hair growing over it.

Mr. YEARSLEY asked whether it was possible that Mr. Cheatle's case was one of the rare hairy polypi which had been described, not a dermoid.

The PRESIDENT (Dr. McBride), in reply, said he was pleased to find that his original impression as to the rarity of the condition was true. He had gone into the literature of the subject, and found that 7 or 8 cases had been described. But in such large works as Parks's "Principles and Practice of Modern Surgery" (1908), and Borst "Lehre von den Geschwülsten" (1902) there were references to dermoids in that situation as if they were of relatively common occurrence: not in so many words, but by implication. One work said that cysts in the mastoid region usually had soft hair. In reference to Mr. Cheatle's case, one would have to find the constituents of true skin in the cyst-wall before it could be called a dermoid.

Mr. JENKINS said he thought there had been some confusion regarding the classification of dermoid cysts in that region. Congenital cysts of the external auditory meatus and outer surface of pinna might be associated with the first branchial cleft, and those situated over the mastoid were probably of the nature of suture dermoids which are fairly common in regard to the whole skull, though uncommon at any particular part of it.

Mr. C. H. FAGGE said there was usually no difficulty in distinguishing microscopically between a dermoid and a sebaceous cyst. The lining epithelium of the latter consisted, at the most, of two or three scanty layers of epithelium, which were distinct from the definite stratified keratinizing epithelium of a dermoid. With regard to dermoids in the neck, upon two of which he had operated in the last fortnight, he would not regard the presence of hairs as pathognomonic, or as necessary for the diagnosis of dermoid. The presence of definite stratified epithelium seemed to him to be enough.

The Position of the Patient after Operations on the Mastoid.

By DAN MCKENZIE, M.D.

It recently occurred to me that, after the mastoid operation, drainage of the large tympano-antro-mastoid space laid open to the external meatus by the operation could be improved by making the patient lie continually upon the affected side rather than by leaving him to choose his own attitude. When left to himself he naturally adopts the most comfortable position, and, refraining from lying on the affected ear, places himself on his back, or, more frequently, on the opposite side, so as to avoid any pressure on the wound. In these positions it is obvious that the cavity formed by the mastoid excavation and the external meatus can drain but imperfectly, while, on the other hand, if the

patient can be kept lying with the affected ear directed downwards, efficient drainage will be perfectly and easily secured. Endeavours to induce patients to maintain this attitude when lying on an ordinary pillow, or upon a circular air-cushion, having proved vain, a pillow was made with a perforation through it, sufficiently spacious to receive the ear and mastoid region without exercising any pressure on the tender parts. On this pillow the patient can lie quite comfortably with the ear downwards, and can be kept in this position as long as he is in bed. The immediate consequences of placing the patient in this position have been that the dressings which drain the meatus become more rapidly soaked with serous discharge than when he lies on the back or on the opposite side, a circumstance which, of itself, seems to suggest that this is the proper attitude. It is clear that if the homolateral position is an advantage after mastoid operations it should also be insisted upon after all operations on the ear, lateral sinus, brain, &c., in which drainage is a matter of importance. Probably the propriety of maintaining the homolateral position after mastoid operations has already occurred to many surgeons, but I have been unable to find any allusion to it in the literature.

DISCUSSION.

Mr. STUART-LOW said that he had seen the pillow in use, and considered that it was a valuable assistance in securing drainage immediately after operation. It was necessary for the patient to maintain the horizontal position, however, while using the pillow, and the pernicious pressure that the head bandage was likely to cause was still possible. To avoid these disadvantages, and at the same time secure perfect drainage, he had invented an aural shield which might sometimes be used at the same time as this pillow. The shield was made in three sizes—the largest was applied immediately after operation, and when in position discharges could pass away freely from the ear into the dressing beneath, and bandage pressure was effectively obviated. Moreover, the patient could be up and about by the third day after operation. He was certain that many narrow meatuses were to be accounted for by bandage pressure. The aural shield having thick rubber edges acted as a Bier's band, and greatly hastened healing.

Mr. HUNTER TOD said he thought the pillow might help to give relief and greater comfort in acute cases, but he did not understand how it could affect the progress of the case, especially with regard to asepsis.

Dr. MILLIGAN asked whether Dr. McKenzie would say what his method of after-treatment was. Did he pack, and, if so, how long did he retain the packing?

Dr. DAN MCKENZIE, in reply, said he had no stereotyped method of after-treatment after mastoid operations; his method depended on the particular case, but he never packed. He was strongly of opinion that the mastoid should always be well drained. The wound after the mastoid operation consisted of two parts: first, the skin incision, which in most instances could be regarded as an ordinary skin wound, for it usually healed by first intention; secondly, there was the very large bone wound, and this it was important to keep well drained and free of stale serum. If the patient lay either on his back, or on the side opposite to that operated upon, the discharges must be retained, an event which could not but retard the healing of the wound. Many otologists treated the mastoid wound as if it were a "clean" surgical wound; they did not change the dressings unless discharges came through or there was pain or fever. In cases where the pillow was used he had noticed that the discharge came through at least every twenty-four hours, sometimes even in twelve hours or less; it was serum pouring out of the bony cavity of the mastoid. So that when the homolateral position was maintained a frequent change of the dressings which drained the meatus was necessary. The homolateral posture, therefore, produced two benefits: it got rid of stale discharges, and it encouraged the transudation of fresh active serum from the blood.

Case of Chronic Middle-Ear Suppuration, with Caries of the Anterior Meatal Wall and Zygoma; history of Local Injury and Syphilis.

By ARTHUR CHEATLE, F.R.C.S.

THE patient was a man, aged 44, suffering from deafness in and discharge from the left ear since boyhood, the cause being unknown. He had syphilis when aged 18. Seventeen weeks before coming to the hospital he had pushed a piece of wood into the ear to allay irritation, without any pain or bleeding. One week later great pain, having its focus immediately above and in front of the ear, and shooting backwards and forwards, came on with a tender swelling, which has increased in size. On examination a firm, exquisitely tender, non-fluctuating swelling was found immediately in front of the tragus, and swelling of the anterior meatal wall, obstructing the view of the deeper parts; the meatus was filled with very offensive discharge; there were no mastoid signs. The parotid swelling increased in size in spite of fomentations, syringing, and antisyphilitic treatment. At the operation the usual dense condition of

the outer antral wall was found, with a small antrum which contained granulations and pus. The anterior meatal wall was entirely carious, the caries extending on to the zygomatic process. There were no cells leading from the middle-ear tract to the zygoma. Several operations had to be performed on superficial extending caries of the zygomatic process. Healing at last occurred under antisymphilitic treatment and the removal of small flakes of bone through a sinus. A plastic operation is necessary to remedy deformity. In my opinion the caries of the meatal wall and of the zygoma was entirely separate from the chronic middle-ear suppuration; perhaps a local wound by the wood became infected by the middle-ear discharge, causing osteitis. The antisymphilitic treatment after the operation was probably of no real benefit.

DISCUSSION.

Dr. A. BRONNER thought the case raised a point of great importance—namely, if a man had had syphilis and then received an injury, whether the injury would determine a local manifestation of syphilis. Some years ago he published cases in relation to the eye, in which typical specific choroiditis occurred in subjects of either acquired or congenital syphilis, after concussion of the eyeball. Such a case as the present might be important from a medico-legal aspect. If a syphilitic patient had an injury and developed a syphilitic manifestation of the injury, was he entitled to compensation?

Mr. WESTMACOTT said there was nothing in the Act about a man suffering entirely from the effects of the injury. If a man liked to employ a workman suffering from a constitutional disease, and who received an injury "arising out of and in the course of the employment," the condition of the workman being aggravated by the constitutional disease, he was entitled to compensation so long as he was unable to follow his employment.

Disease of External Auditory Meatus.

By W. H. KELSON, M.D.

THE patient, a girl aged 10, with white deposit in both external auditory meatuses, was shown at the last meeting. Subsequent history of the case: A few days after the meeting the patient was examined under an anæsthetic, and the membrana tympani on each side was found to be intact. Cultures showed the presence of *Staphylococcus aureus*, but the Klebs-Löffler bacillus could not be found, nor did smears show presence of any mycelium. The ears were treated with glycerine of carbolic acid, and the patient was discharged well in ten days.

DISCUSSION.

The PRESIDENT said the case was of considerable interest. It turned out to be one of otitis externa; not a very common condition.

Mr. C. E. WEST asked whether the statement that the Klebs-Löffler bacillus could not be found implied that there were no diphtheroid bacilli of any sort in the cultures.

Mr. KELSON replied that no diphtheroid organisms were found in connexion with the case.

**Sections of the Human Cochlea through the Organ of Corti,
showing Beaded Nerve-fibril traversing the Tunnel of
Corti, and Cell-fibrillæ projecting from the Hair-cells.**

By SYDNEY SCOTT, M.S.

METHOD of preparation¹: The petrous bone of a child was removed and fixed in Flemming's "strong" solution² within one and a quarter hours of death; the superior and posterior semicircular canal and the cochlea were opened to permit more rapid penetration of the fixative, and the stapes was partly displaced. After sixteen hours the bone was transferred to running water for one and a half hours and was left in standing water for three quarters of an hour. Decalcification was obtained by means of nitric acid and phloroglucin solution,³ containing 15 per cent. nitric acid and $\frac{2}{3}$ per cent. phloroglucin in water; 300 c.c. of the decalcifying reagent were used, and the process was complete within seventy-two hours. After the removal of the excess of acid by washing in water, the mass was dehydrated in alcohol, cleared in xylol and imbedded in paraffin

¹ Modification of a method previously described, see *Proc. Roy. Soc. Med.*, 1908, i (Otol. Sect.), p. 19; also *Proc. Anat. Soc. Great Britain and Ireland*, March, 1908, p. 7 (*Journ. Anat. and Physiol.*, Lond., 1908, xliii).

² Flemming's "strong" solution prepared by adding one part of a mixture of 2 per cent. osmic acid in 1 per cent. chromic acid to four parts of the following solution: 1 per cent. chromic acid, eleven parts; aqua pura, four parts; concentrated acetic acid, one part.

³ Instead of the usually described method of preparing the nitric acid and phloroglucin solution, I find that it is more conveniently prepared by adding very slowly the phloroglucin crystals to the pure nitric acid, contained in a large thin glass beaker, surrounded by water at a temperature of about 15° C. to control the temperature of the mixture. Ebullition of the solution is very apt to occur, and must not be permitted on account of the decomposition of the acid with wastage of material and danger of nitrous fumes. On the other hand it is essential that all the phloroglucin should be dissolved, otherwise the bone rapidly disintegrates.

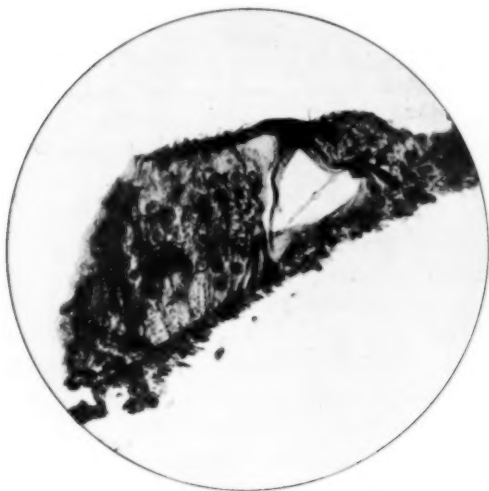


FIG. 1.

Section of the human organ of Corti, approximately radial and approximately vertical, 6 microns thick. A beaded nerve-fibril is seen crossing the tunnel of Corti. ($\times 320$; microphotograph.)



FIG. 2.

True vertical and approximately radial section of human organ of Corti. (Microphotograph, focussed on to the neck of one of the hair-cells; $\times 270$.)

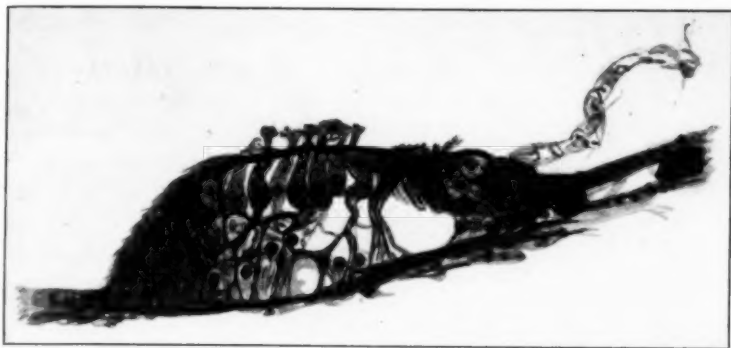


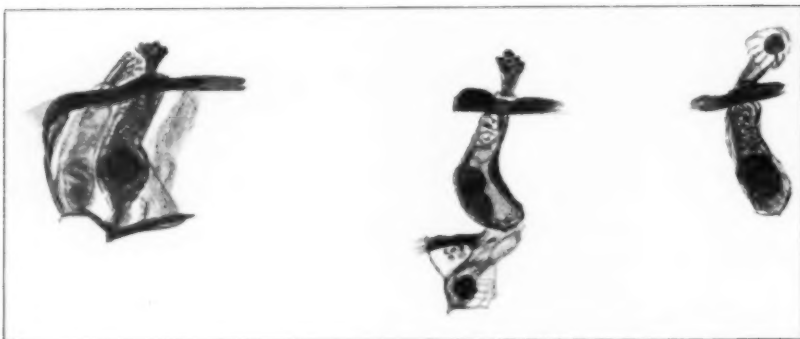
FIG. 3.

Drawing of the same section (that is fig. 2) with camera lucida by
Mr. M. H. Lapidge. ($\times 300$.)



FIG. 4.

Microphotograph of same specimen (see figs. 2 and 3). The nature of the
black disc-like object is interpreted in fig. 5, C. ($\times 1,000$.)



A

B

C

FIG. 5.

Hair-cells seen in the same section (see figs. 2, 3, and 4). *A*, Two adjacent cells in the outer part of the organ; *B*, vertical section of a middle cell with cell-fibrillae traceable into the body of the cell; *C*, body of a hair-cell with the fibrillae of an adjacent cell showing the expansion of the fibrillae (cf., fig. 4). (Drawings by Mr. M. H. Lapidge with camera lucida; $\times 600$.)

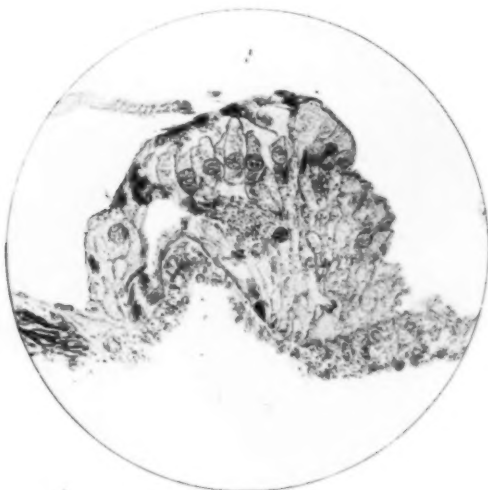


FIG. 6.

Section of human organ of Corti, very thin (3 microns thick). To illustrate possible erroneous impressions, the section not being perfectly perpendicular or perfectly radial, the cell-bodies are cut obliquely and only approximately radial, so that 5 nuclei appear, all in focus. Parts of the cell-fibrillae of adjacent cells are recognizable. The striations of the membrana tectoria can be seen. The membrana basilaris has become bent where it forms the floor of the tunnel of Corti. The termination of the medullated nerve-sheaths is seen on one side of the section. (Microphotograph; $\times 320$.)

wax. Horizontal sections, 3, 4, 5 and 6 microns thick, were cut from below upwards through semicircular canals, vestibule and cochlea, with a Leitz microtome, a new (1908) model, sledge pattern cutting on the flat. Each section was mounted and examined before being discarded or permanently preserved. The only sections of the cochlea which were in an absolutely vertical plane of Corti's organ consisted of a small group on a level with the upper margin of the fenestra ovalis; the sections above and below this small group were all cut at varying degrees of obliquity, and therefore do not show the essential structures of the organ in the same way as the specimens illustrated.

I am greatly indebted to Dr. Albert Norman for the microphotographs which he took from the sections themselves. The illustrations are pure photographs and have not been reconstructed in any way.

DISCUSSION.

Dr. URBAN PRITCHARD said the demonstration had been very interesting to him. The slides showed what he had generally found—namely, that the hair-cells had generally four hairs in a row, and the rows went with the spiral. Yet in some books these rows were described as going against the spiral. In the human cochlea the number of rows of hair-cells increased as one got up the spiral; he had found three, four, and five. He did not know whether Mr. Scott found more. With regard to the nerve-filament which was shown passing across the tunnel, he had a number of specimens from the lower animals showing it, but none from the human subject. The membrana tectoria was merely a mucoid layer; there was no definite structure, and it was homologous to the mucoid layer, which contained the otoliths in the vestibule. With regard to the theory as to the vibration of the membrana basilaris being of importance in appreciating pitch, if that were so, one would not find any part of the organ of Corti without a membrana basilaris, whereas in the upper part of the cochlea of birds there was a part of the organ of Corti without any membrana basilaris at all.

Dr. MILLIGAN said Mr. Scott was to be very sincerely congratulated on the lovely sections which he had exhibited. They must have entailed great labour, and he hoped it would be possible to have reproductions of them in the *Proceedings*. He asked how soon after death Mr. Scott removed the temporal bone.

The PRESIDENT asked whether Mr. Scott had paid attention to the relation of the membrana tectoria, and whether he had seen a paper by Shambaugh, of Chicago, in which it was said that the tectorial membrane vibrated and increased in length towards the apex. He thought a strong case was there made out in favour of it vibrating. He (Dr. McBride) thought there was a striation in the membrana tectoria which indicated definite structure.

Mr. SCOTT, in reply, said he could not tell whether in the upper parts of the human cochlea there were five outer hair-cells to the row or not, as the sections shown were from the middle coil. In his earlier sections he had found rows of five and more outer hair-cells in various parts of the cochlea, but with improved technique, thereby obtaining thinner and accurately radial sections, he found only three and four cells in a row, which agreed with Retzius's observations. To those who, like Gottstein, had held that there were sometimes rows of five outer hair-cells in the human cochlea, he would suggest the possible objection that the sections were perhaps not in a perfectly radial plane. With regard to the nerve-fibrils across the canal of Corti, he understood that in the lower animals in which the organ of Corti was present these fibrils could be demonstrated, but these specimens were the first which he had seen in which the fibrils could be recognized in the human subject. Mr. Scott stated that the arrangement of the fibrillæ of the hair-cells was different from that which he believed had hitherto been described. Instead of being arranged in parallel rows of short, straight filaments, as figured by Retzius and others, Mr. Scott's interpretation of the section shown was that the fibrillæ of Corti's cells began within the cell about the junction of the middle with the upper third, and that they emerged from the upper opening of the cell as a diverging spray of filaments which curled over slightly at their extremities. In reply to the President, he had not yet made a complete comparative study of his sections of the membrana tectoria, but he had noticed this membrane was present where there were hair-cells, and that its structure and appearance was in favour of Shambaugh's views regarding the functions of the membrane. He could not say whether the membrana tectoria was composed of mucoid substance or not, but if so it differed from any structure composed of mucoid material with which he was acquainted, for the membrana was uniform in contour and structure; in places definite vertical striations, which were very regular and very close together, could be observed. In answer to Dr. Milligan, the present specimens were obtained one hour and fourteen minutes after death.

Four Cases of Carcinoma of the External Meatus.

By C. E. WEST, F.R.C.S.

THERE had been a series of seven of these cases in the Aural Department of St. Bartholomew's Hospital during the last two years, and in addition one of round-celled sarcoma of the pinna. Mr. West would venture to suggest that malignant disease was a more common condition than was generally accepted, and that a thorough search for cases would probably show that they were not really rare. He thought

that there was good evidence in at least six of these cases of the origin of the disease in the meatus, and in three of them there was undoubtedly evidence that the discharge was secondary to the presence of the growth. This seemed to commence in the floor of the osseous meatus, and in three had led to destruction and secondary necrosis of the lower part of the tympanic plate. In all, the growth was a squamous-celled carcinoma, and all had been submitted to the verdict of skilled pathologists. Lymphatic spread seemed to be first to the preauricular lymphatic glands, then to the upper deep cervical glands. In one of the cases shown there was a large malignant mass in the root of the tongue on the side affected, presumably in the lymphoid tissue in that situation, and secondary to involvement of the cervical glands. One case had died of involvement of the dura mater and brain; for this case he was indebted to Mr. Sydney Scott.

In operating on these cases the principle followed had been to completely remove the meatus, both cartilaginous and bony, preserving the pinna. In addition the tissues in front of the ear were removed down to the parotid fascia, so as to include the preauricular glands and their afferent lymphatics, and freely behind and below. This necessitated the removal of the root of the styloid process in several of the cases. A sweeping radical operation was then carried out and the disease was followed in any extensions. In one case (shown) the whole of the facial nerve from the geniculate ganglion to the stylomastoid foramen had been dissected out in order that infiltrated bone on its deep aspect in the sinus tympanicus might be reached; that patient had complete control of the face on the side of operation. After-treatment might be either with open wound behind or on the ordinary lines of a radical operation. It was advantageous to graft the cavity.

As to results: Of six operations one case had died of sepsis spreading into carcinomatous brain tissue; this case was initially a hopeless one. One case had been relieved for six months of the local disease and of pain; he had extensions which at the time were irremovable, and no operation was attempted on them. Four cases were so far free from recurrence; the earliest of these was operated on in June, 1907.

He suggested, finally, that some of these cases may have been missed as cases of brain abscess or as cases of carcinoma of lymphatic glands with unknown primary focus, or as cases of carcinoma of the parotid.

DISCUSSION.

Dr. DUNDAS GRANT said Mr. West was to be congratulated on his skill and boldness in regard to the cases, and he cordially agreed with what he had said with regard to diagnosis. He had himself seen several cases, and the characteristic features at the early stage, when the disease in the meatus was comparatively inconspicuous, was the intense pain which extended over wide areas, and was quite out of proportion to the amount of change in the ear. The ear condition looked like simple granulations, and casual observation would not give a hint of their seriousness, as revealed by the microscope, but one thing which impressed him very much was the amount of exposed bone. When the granulations were touched with a probe in order to find whether they led into a fistula extending to the mastoid cells there was found to be an enormous area of exposed bone. This occurring with severe pain in an adult should arouse a strong suspicion of its malignant nature. He was convinced that a bolder surgery than had hitherto been practised was justifiable if the search for the condition was made sufficiently early.

Mr. SYDNEY SCOTT desired to emphasize the necessity of pathological evidence by obtaining sections of the growth before deciding whether it was malignant or not. This had been done at the time of the operations in accordance with Mr. Lockwood's plan,¹ introduced to St. Bartholomew's Hospital nine years ago, of obtaining the services of an experienced pathologist in the theatre at the time of the operation, so that in a few minutes one could be certain of the nature of the disease. Subsequently permanent sections of the growth were required for reference. He had seen all the cases in this series, and was convinced of their nature by the appearances of the disease, confirmed by the microscopical characters of the growths removed.

The PRESIDENT (Dr. McBride) said he thought Mr. West's experience must have been quite exceptional, because during the twenty years he (Dr. McBride) was attached to Edinburgh Infirmary, and in his private practice, he had seen only three cases. He gathered from the work of others that malignant disease of the ear appeared apart from malignant disease of the pinna, but in two forms. In one form there was chronic suppuration of the middle ear, with granulations appearing. The less common form seemed to be that described by Mr. West; he had seen one case where there was a small painful point in the ear, and the continuance of the pain made him suspect malignancy, which the microscope confirmed. He removed freely, but did not lay bare the anterior glands. He found the mastoid gland enlarged, and removed that. He thought Mr. West must have happened by chance on a series of those cases.

¹ Vide "The Immediate Microscopical Diagnosis of Tumours during the Course of Operation," *Brit. Med. Journ.*, 1907, i, p. 127.

Mr. WEST, in reply, admitted that it was a remarkable series, but when such a series extended over two years, probably the coincident element became small. He believed the reason the cases had been found so frequent was that they had been looked for carefully with scrupulous suspiciousness. One was not diagnosed as carcinoma until the rapid recurrence of the disease in the cavity excited suspicions of malignancy. He believed many cases went on to a hopeless condition without their seriousness being recognized. He remembered a man with a huge mass of epitheliomatous glands in the upper part of the triangles of the neck; he searched everywhere for the primary growth except the ear and did not find it. Probably this was a case of missed carcinoma of the meatus.

Notes of a Case of Chronic Osteomyelitis of the Skull, the Result of Mastoid Disease (with Specimen of Calvarium).

By HUNTER TOD, F.R.C.S.

THE patient, a woman aged 53, was admitted to the London Hospital on April 22, 1908, suffering from a purulent discharge from the right ear, which had existed for two years. For two months previous to admission there had been constant pain behind the ear, accompanied by headache and pain in the eye. There was an obvious abscess over the mastoid process. The left ear was normal. On performing the mastoid operation on the right side a large quantity of pus was found beneath the periosteum, and the bone above and behind the mastoid process was necrosed over an area the size of the palm of the hand. Its removal caused extensive exposure of the dura mater covering the lateral sinus and also of the lower portion of the temporo-sphenoidal lobe and cerebellum. The dura mater was thickened and covered with granulations. There was also extensive necrosis of the posterior wall of the auditory canal. The wound was left open and packed posteriorly. After operation there was complete relief from the head symptoms, and the temperature remained normal throughout. The patient was dismissed to the out-patient department after three weeks, with an apparently healthy granulating wound. After two months the headaches returned. Meanwhile the wound had only partially healed, and from its upper posterior margin a further piece of

necrosed bone was removed. In October, 1908, the patient was readmitted to the hospital with a puffy swelling over the parietal region of the skull on the affected side, which was extremely tender on palpation. An incision was made over this part, and the bone found to be carious. From this date onwards the headaches continued, and there appeared to be a spreading cellulitis extending over the whole of the skull, requiring multiple incisions. The patient was transferred to my care in December, 1908. On the right side (the ear originally affected) there were no active symptoms of disease. There was, however, considerable discharge from the left ear, but no swelling nor tenderness over the mastoid region. There was extreme deafness. The patient complained continually of gnawing pain in the head. Paralysis of the left facial nerve had existed for ten days. The temperature was only occasionally raised above normal; pulse 76 and regular. There were slight attacks of vomiting at irregular intervals. The diagnosis was that of chronic osteomyelitis, and it was presumed that the left ear had become infected by direct extension through the bone from the opposite side. Although the prognosis seemed hopeless, I intended to operate on the left side, hoping to relieve the head symptoms, but before this could be done the patient suddenly collapsed and died.

Post-mortem Report.—"Fairly well nourished. Scar behind right ear. Sinuses in scalp on each parietal eminence leading to bare bone. Scalp is very thick and oedematous and strongly adherent to skull all over. On stripping off the pericranium the skull bones are seen to be irregularly worm-eaten; the sunken dark red sinuous areas do not extend quite through the outer table, but in the region of the left external fontanelle is a considerable depression extending almost through the skull. At the anterior fontanelle the bone is very thin. At the posterior angle of the right parietal there is a deep depression, in the floor of which is no bone, but only periosteum and dura mater fused together. Similar boneless depressions are to be seen above and behind the right mastoid, which is practically separated from the skull. On removing the skull-cap the dura is seen to be adherent to the bone and to the periosteum at those spots where the osseous tissue is completely absent. There is no excess of cerebro-spinal fluid beneath the membrane. Beneath the tentorium covering the whole posterior fossa and base of brain is thick green pus, which also fills the left internal meatus and extends down into the spinal sheath. The bone of the tegmen tympani is red, soft, and granular. Both middle ears contain soft, cheesy matter. On splitting the left internal ear no pus is found in the vestibule, but the superior semicircular

canal is lost in a large cavity filled with granulation tissue. Both lateral ventricles of the brain are considerably dilated and filled with clear fluid, but the brain substance itself is pale and firm and contains no abscess."

DISCUSSION.

The PRESIDENT (Dr. McBride) said he was acquainted with some reports of cases where that had occurred after frontal sinus operations, and he had had experience of one case. He had never seen it in connexion with mastoid operations, and did not think many such had been described.

Mr. C. E. WEST said he mentioned one similar case at a recent meeting. There had been a radical operation, and there was a spreading swelling which was due to periostitis and osteitis of the skull bones, and spread over the whole calvarium. The other ear suppurated violently, and the patient died with an undiscovered frontal abscess nine months after the mastoid operation. He asked whether Mr. Tod could throw any light on the bacteriology. In his case the chronic abscesses formed in an enormously thickened white induration of periosteum and contained glairy purulent fluid; when fluid was withdrawn by needle nothing grew in it except a streptothrix, which grew in blood broth, and which could always be demonstrated in films of the pus. It was finally found in the frontal abscess.

Dr. DONELAN said the conditions favouring chronic osteomyelitis in the human subject were fortunately of rare occurrence. The loose arrangement of the diploe was the most important contributing factor to the spread of infection. It seemed to him it had some analogy with a condition common in many lower animals, the extreme type occurring in the elephant, in which the skull was practically double.

Mr. SYDNEY SCOTT asked for information regarding the infection of the left middle ear, the presence of granulations in the semicircular canals and pus in the internal auditory meatus on the same side; whether the infection arose while the patient was under Mr. Tod's care, and whether Mr. Tod was of the opinion that the infection of the internal ear was secondary to or the cause of the meningitis.

Mr. TOD, in reply, said he apologized for the notes being somewhat incomplete, but he did not see the beginning nor the end of the case. The condition was first diagnosed by his house-surgeon as an acute mastoid abscess, who for this reason was permitted to perform the operation. The patient was readmitted in October, 1908, under a surgical colleague. The bone was never freely exposed, but the inflammatory swellings were merely incised and fomentations afterwards applied. In answer to Mr. Scott, Mr. Tod said that he presumed that the left ear became infected through the bone by gradual

extension of the disease from the opposite side, and probably the middle and internal ear became infected at the same time. Unfortunately, no bacteriological investigations were carried out. The chief subjective symptom was a continual intense gnawing pain in the head. The temperature, however, was never higher than 99° F., in which point it differed from other reported cases of osteomyelitis.

Otological Section.

March 6, 1909.

Dr. PETER McBRIDE, President of the Section, in the Chair.

The Problem of Vertigo : some new Data obtained in a Research into the Functions of the Semicircular Canals in Relation to Movements of the Eyeball in the Human Subject.

By SYDNEY SCOTT, M.S.

CONTENTS.

- (1) Introduction, chiefly historical.
- (2) Scope and general arrangement of subject-matter.
- (3) Definition and varieties of vertigo and nystagmus.
- (4) Types of labyrinthine nystagmus. Nomenclature.
- (5) Tables of data obtained by direct observations of the effects of changes of temperature of the ear, and of rotation on eye-movement.
- (6) The physical effects of relative heat and cold on the semicircular canal apparatus.
- (7) The physical effects of rotation on the semicircular canal apparatus.
- (8) Comparison of the hypotheses of convection and momentum currents.
- (9) Some theoretical data of rotation which have a bearing on technique.
- (10) The theory of the stimulus producing labyrinthine nystagmus.
- (11) Application of the principles laid down to explain the nystagmus associated with labyrinthine fistulæ (extra-ampullary lesions).
- (12) The relations of the semicircular canals to normal ocular fixation.

- (13) Observations on ocular fixation during immobilization of the head.
- (14) Observations on ocular fixation during head movements.
- (15) The synthesis of labyrinthine nystagmus. Theory of its mechanism.
- (16) Crum-Brown theory of paired canals.
- (17) The effect of removal of both sets of semicircular canals (bilateral ablation). Absence of labyrinthine nystagmus.
- (18) The effect of destruction by disease, or removal by operation of one set of semicircular canals (unilateral ablation). Explanation of the occasional spontaneous nystagmus associated therewith.
- (19) Comparison of the spontaneous nystagmus of unilateral ablation with the nystagmus associated with labyrinthine fistulæ and intact membranous labyrinth.
- (20) A new hypothesis to explain unilateral ablation nystagmus.
- (21) Conclusions.

WITHIN the last two or three years considerable advances have been made in our clinical knowledge of nystagmus. The phenomena of certain varieties of eye-movement were noted, and explanations were offered, by Purkinje, Flourens, Mach, Breuer, Cyon, Crum-Brown, and more recently by Lee and others, in connexion with experimental methods of stimulating the labyrinth of vertebrates, including man. Professor Robert Bárány, of Vienna, realized the value of systematic examination of eye-movement in human clinical otology. His procedures of examination cannot be too well known, for many of his observations have been confirmed by other investigators. I think we may regard Bárány's observations and those of his confrère, Dr. Gustav Alexander,¹ as representing the most valuable contributions to clinical otology which have been made in recent years.

I am not aware that any very complete or satisfactory explanation of nystagmus has yet been described, and I venture to bring this subject forward as an independent study of the human membranous labyrinth, both in health and disease, with numerous observations upon nystagmus in many individuals, in a number of which opportunities for ascertaining the state of the labyrinth have subsequently arisen. It so happens that I have met with certain data which I cannot find have been so far noticed, and the significance of which appears to me to justify my submitting them for the consideration and criticism of others.

¹ *Pflüger's Arch. f. gesamt. Physiol.* (Pflüger), Bonn. 1902, lxxxix, p. 465; *Zeitschr. f. Ohrenheilk.*, Wiesb., 1908, lvi, p. 138.

INTRODUCTION, CHIEFLY HISTORICAL.

Purkinje, in 1820, described the phenomena of rotation and the sense of vertigo, and noted that during continued rotation of the body about a vertical axis the eyes at first remained fixed on external objects, then followed the movements of the head in jerks, then, as rotation continued, the eye appeared to lag behind, and, lastly, to move at the same velocity as the head. On cessation of rotation eye-movements in jerks were renewed. Purkinje assumed these movements were provoked by lateral pressure of the brain within the cranium.

In 1842 Flourens, by direct experiment, showed that the sense of equilibrium was related to the semicircular canals of the labyrinth. Very many other investigators confirmed this view. Although a large number of facts have been collected by different observers at different times, the precise relation of equilibrium to the semicircular canals has not been made perfectly clear.

In 1869 Löwenberg tried the effects of chemical and mechanical irritants to the ampullæ of the semicircular canals of birds, and was led to conclude that eye-movements which were produced were reflex, and that the centres for the action were in the optic lobes.

Goltz formulated the essential conditions of the reflex mechanism from Flourens's data, and propounded the hypothesis that the endolymph exerted pressure on the walls of the ampulla during resting states, thus irritating the nerve-endings, setting up impulses which varied with resting positions of the head.

In 1878 Cyon made numerous experiments by dividing different semicircular canals in pigeons, and described certain inco-ordinate movements which passed off after eight to ten days, and which we should probably now ascribe to irritating lesions as met with in labyrinthine fistulæ. He found that stimulation of the semicircular canals in birds excited oscillatory spasms of the eye-muscles, which he noticed to be at the rate of twenty to 150 jerking movements per minute.

In 1874 and 1875 Mach and Breuer made some valuable contributions to the subject. Mach showed that Purkinje's phenomena of vertigo and nystagmus should be ascribed to the semicircular canals, after he had made an elaborate investigation of all the organs which might conceivably give rise to such phenomena. Breuer made a prolonged research upon the results of rotation, thermal and galvanic stimulation. According to Frederick Lee, Breuer's is the most elaborate

work which has been carried out on the functions of the semicircular canals. Mach agreed with Breuer's suggestion that the ocular movements were reflex and excited by impressions in the ampullæ; that such movements were normally compensatory to aid vision, and that they normally occur in opposite directions to movements of the head.

In 1874 Crum-Brown independently investigated the results of rotation in the human subject, and enunciated a dynamic theory of the stimulus similar to that of Mach and Breuer, which is opposed to that of Goltz's static hypothesis. Mach attributed the stimulus to variations of pressure in the ampullæ rather than to actual flow of fluids through these minute canals. Crum-Brown was of the opinion that movement or variation of pressure occurred not only in the endolymph, but also in the walls of the membranous canals and surrounding perilymph. He regarded the two labyrinths as essentially one organ, the two horizontal canals corresponding, the ^{right}_{left} superior canal corresponding with the ^{left}_{right} posterior canal.

In 1892 Lee made experiments in skates and dog-fishes, and observed the eye- and fin-movements during rotation, after section of the acoustic nerve, on one and both sides, after section and stimulation of each semicircular canal, and of each ampullary nerve, in turn. Lee concluded "that each ampulla has a principal and a subordinate function; that if the function of one ampulla regulates movements of the eyeball in one direction the subordinate function is to produce ocular movement in the opposite direction. Gentle pressure with a needle on the anterior ampulla caused the eyeball of the same side to roll slightly downwards, and that of the opposite side slightly upwards. This is the subordinate function. A stronger pressure causes the eyeball of the same side to roll strongly upwards, and that of the opposite side to roll strongly downwards—this is the principal function." Lee attributes the normal stimulating agent of each end-organ in the ampulla to "the inertia of the endolymph through which the hair-cells of the crista are dragged" (p. 345). He noted that the loss of a single canal does not interfere with the animal's equilibrium, but the loss of the functionally paired canals removes compensatory power. The immediate effect of the loss of all three semicircular canals on one side was to weaken, not to destroy, the power of compensation. With the loss of all six canals the compensatory power for all planes disappears, together with rotational equilibrium (p. 346).

Section of one acoustic nerve produced the same results as the destruction of the three semicircular canals on the same side, and

interferes only slightly with statical and dynamical equilibrium, without abolishing power of compensatory movement. "Section of both acoustic nerves destroys much more completely the sense of both static and dynamic equilibrium, even to the extent of doing away with compensatory power." By weak galvanic stimulation of the central end of the cut acoustic nerve, he found he could pick out different groups of nerve-fibres, with the electrodes, stimulation of which gave rise to the same movements as those obtained by stimulation of the ampulla to which the particular nerve-group corresponded. When the whole nerve was stimulated the resultant reflexes corresponded to the components of the whole nerve, and the direction of ocular deviation was the reverse of that which appeared to follow section. Unfortunately, Lees does not distinguish anodal and kathodal stimuli, and he does not appear to have observed any difference in the direction of eye-movement attributable to the change in direction of the current. Nor does he state the ultimate effects of his experiments on ocular movements.

Note.—For the foregoing historical notes I am indebted to Albert Gray and McKendrick's article in Schäfer's "Text-book of Physiology," as well as to the following monographs and papers:—

- BÁRÁNY. "On Labyrinthine Nystagmus," digest by THOMAS GUTHRIE, *Brain*, Lond., 1906, xxix, p. 383.
 CRUM BROWN. "On the Sense of Rotation and the Anatomy and Physiology of the Semicircular Canals of the Internal Ear," *Journ. Anat. and Phys.*, 1874, Lond., viii, p. 327.
 LEE, FREDERICK S. "A Study of the Sense of Equilibrium in Fishes," *Journ. of Phys.*, Camb., 1893, xv, p. 311.
 STIRLING. Digest of "The Report on Physiology," contained in *Journ. Anat. and Phys.*, 1874, viii, p. 400, and also 1875-6, x, p. 634.

Other references quoted by Gray and McKendrick, and by Lee:—

- BREUER. *Med. Jahrb.*, Wien, 1874-75, pp. 72, 87.
 DE CYON. "Recherches expérimentales sur les fonctions des canaux sémicirculaires," *Thèse de Paris*, 1878.
 GOLTZ. *Arch. f. d. ges. Phys.* (Pflüger), Bonn, 1870, iii, p. 172.
 LOWENBERG (quoted by STIRLING). *Arch. f. Augen u. Ohrenh.*, Carlsruhe, 1869, iii, 1 Abth., p. 1.
 MACH U. KESSEL. *Sitzungsab. d. k. Akad. d. Wissensch.*, Wien, 1874, lxix, 3 Abth., p. 221.
 PURKINJE. *Med. Jahrb.*, Wien, 1820, vi, 2 Stück, p. 79.

SCOPE AND GENERAL ARRANGEMENT OF SUBJECT-MATTER.

(1) The varieties of nystagmus in the human subject which I have studied will be described, together with the conditions under which the observations were made.

(2) An explanation of the essential nature of the stimulus which provokes nystagmus will be adduced by considering the structural

anatomy of the utricle and semicircular canals, and the physical agents which set up nystagmus.

(3) Various conditions under which nystagmus has been observed will be compared and discussed.

(4) The functions of the semicircular canals in relation to normal ocular fixation will be considered, with the deductions based upon preceding data.

(5) I shall then describe some recent experiments on eye-movements apart from the semicircular canals, and shall endeavour to analyse and resolve nystagmus into two comparatively simple reflexes.

(6) Lastly, I shall present some data, which in the light of these observations seem to justify a new hypothesis explaining nystagmus which sometimes follows destruction of all the semicircular canals on one side in the human subject, and which hypothesis may perhaps explain some obscure forms of nystagmus and vertigo connected with certain intracranial lesions and with certain disorders of the vascular system. Owing to the limitation of my field of research I am not able to discuss the situation of our faculty of orientation, or the varieties of subjective sensations of the state of vertigo, or the symptomatology of cerebellar tumours. These topics have been brought to our direct notice in recent years by Sir Victor Horsley and Dr. Risien Russell.¹

DEFINITIONS AND VARIETIES OF VERTIGO AND NYSTAGMUS.

A person is said to experience giddiness when he has the subjective sensation of movement about an axis in some direction in space, whether he be really moving or not; this sensation may be accompanied or overpowered by a sensation that surrounding objects are moving in some definite direction when in reality they are at rest. When the sensation of giddiness is sudden and severe it may be accompanied by reeling, and we may reserve for this state the name vertigo. When the giddiness is very slight and transient, and when its directional characters are indefinite, we may call it dizziness. In spite of these distinctions we find it convenient to employ the terms vertigo and giddiness synonymously. All these forms of giddiness may be associated with more or less obvious nystagmus. On the other hand, true vertigo may occur without any recognizable nystagmus.

The following are the main varieties of nystagmus (*νυδάζω*, to nod) of which I shall speak. One group includes those varieties the most

¹ *Trans. Otol. Soc. U.K.*, Lond., 1905, vi, pp. 72-79.

noticeable feature of which is that the velocity of movement of the eyeball in one direction is equal to the velocity of movement in the opposite direction, so that a series of to-and-fro excursive movements equal in amplitude and period can be recognized. This variety occurs especially in persons with certain kinds of defective vision, particularly due to opacities in the cornea or other media which interfere with the axis of most distinct vision. Other forms of nystagmus in which the movements may or may not be equal in period are unconnected with the above visual defects, but are met with in association with fine or gross tremulous movements of muscles in other parts of the body. Whether certain forms of nystagmus met with in some cases of insular sclerosis should be included in this class or not I am not competent to say. Nor is it within my sphere to enter into a discussion as to the nature of the above forms of eye-movement. I have had no experience of miner's nystagmus, nor of the nystagmus associated with head-nodding in children. These forms have been mentioned to clear the ground for an important variety with which I am especially concerned, and which includes all kinds of nystagmus produced by direct and excessive stimulation of the labyrinth. It will be my endeavour to give reasons why we should include in this class the various types of nystagmus which sometimes arise as the result of destruction of all the end-organs in one labyrinth. I shall show good reasons for the belief that a labyrinth which is the seat of diffuse infection is functionless; that a labyrinth is functionless when the vestibule and ampullæ contain granulation tissue; that it is rendered functionless by opening the vestibule and curetting its contents away; that section of the auditory nerve renders the labyrinth with which it was connected functionless, and I shall ask to be allowed to presume that when one auditory nerve is destroyed by an intracranial lesion the labyrinth on that side ceases to functionate, even though the organ may not be visibly changed.

It will be necessary to enter into a detailed description of the types of ocular movement comprising so-called labyrinthine nystagmus.

TYPES OF LABYRINTHINE NYSTAGMUS.—NOMENCLATURE.

According to the form of excursive movement we shall find it convenient to speak of three main types—namely, horizontal, vertical, and rotatory nystagmus.

By the term horizontal nystagmus one understands that the plane of oscillation is horizontal, not in relation to space, but in relation to the normal horizontal plane of the orbit.

Vertical nystagmus similarly has reference to the vertical plane of the orbit.

Rotatory nystagmus is more conveniently considered in relation to rotatory oscillation around the antero-posterior axis of the eyeball. It is usual to speak of the more rapid of the two oppositely directed movements as the major movement. I shall later show reasons for regarding the slower movement as the primary movement, or movement of "reflex compensatory deviation" (of Mach and Breuer) set up by labyrinthine impulses, the rapid movement, or major movement, as secondary, and due to the attentive reflex movement of pursuit. The term pursuit is taken from Dodge, who in connexion with other work on the eye has made use of the terms "reactions of pursuit."¹

Labyrinthine nystagmus is most intense when the visual axes are directed towards a certain point on the limits of the binocular field of vision. The direction of the rapid or major movement is towards this point of maximum intensity. The point of maximum intensity varies with the type of nystagmus. In horizontal nystagmus the point of maximum intensity is found by attentive deviation of the eyes to the right or left, and we speak of right or left horizontal nystagmus. Similarly, the point of maximum intensity for vertical nystagmus is found by attentive deviation towards the upper or lower limits of the visual field, and we speak of vertical nystagmus upwards or vertical nystagmus downwards.

As regards rotatory nystagmus, the maximum intensity of rotatory oscillation is less obvious owing to the short range of possible movement, as in the human subject. The rotation of the eyeball may be more rapid either clockwise or counter-clockwise; and when the movements are slight they occur on attentive deviation most commonly to the right or to the left, but in some cases the deviation of attempted fixation need not be to the extreme margin of the binocular field, nor need the deviation be purely horizontal. During vertical deviation and attentive fixation, either upwards or downwards, rotatory nystagmus may be present.

Various combinations of these types occur, such as oblique nystagmus, which may be regarded to be composed of vertical and horizontal types in the case of labyrinthine nystagmus. We also frequently meet with combined horizontal and rotatory nystagmus. Of all these types by far the most difficult to recognize is rotatory nystagmus. I have found it well to watch the motion of the conjunctival vessels near the cornea, by

¹ See *Brain*, Lond., 1908, xxxi, p. 451.

which one can more readily recognize the slighter forms of rotatory oscillation.

The anatomical data represent a considerable part of my research, upon which all the deductions which I have made have been based

TABLES SHOWING DETAILED RESULTS OF RESEARCH.

The following tables indicate the various methods by which I have obtained the three main types of labyrinthine nystagmus which have been described:—

TABLE I.—ROTATORY (COUNTER-CLOCKWISE) NYSTAGMUS OBSERVABLE DURING ATTENTIVE DEVIATION AND FIXATION OF THE EYES TO THE RIGHT.

By Thermal Methods.¹

Cold water irrigation	...	Left ear	...	Head erect
Hot " "	...	Right "	...	" "
" " "	...	Left "	...	" inverted
Cold " "	...	Right "	...	" "

By Rotation around a Vertical Axis.¹

Rotation counter-clockwise	...	Face directed downwards
" clockwise	...	" " upwards

¹ For an account of technique adopted by Bárány see Thomas Guthrie's digest, *Brain*, 1906, p. 383; also Mackenzie's abridged translation in *Journal of Laryngology, Rhinology and Otology*, xxiv, No. 2. The present series of observations was carried out by methods evolved independently of Bárány's, though essentially similar. The conclusions were also formulated before the writer studied the results of Breuer's investigations.

TABLE II.—ROTATORY (CLOCKWISE) NYSTAGMUS OBSERVABLE DURING ATTENTIVE DEVIATION AND FIXATION OF THE EYES TO THE LEFT.

By Thermal Methods.

Cold water irrigation	...	Right ear	...	Head erect
Hot " "	...	Left "	...	" "
" " "	...	Right "	...	" inverted
Cold " "	...	Left "	...	" "

By Rotation around a Vertical Axis.

Rotation clockwise	...	Face directed downwards
" counter-clockwise	...	" " upwards

Note.—The effect of rotation on the eye-movements depends upon the angular velocity and tangential speed. A certain minimum velocity is necessary, and this varies somewhat in different persons. Given the appropriate angular velocity and appropriate tangential speed one finds that rotation will induce the same type of nystagmus with the same directional characters whether both labyrinths are functional or whether one labyrinth had become defective (see also p. 54).

TABLE III.—HORIZONTAL NYSTAGMUS OBSERVABLE DURING ATTENTIVE DEVIATION AND FIXATION OF THE EYES TO THE RIGHT.

By Thermal Methods.

Cold water irrigation	...	Right ear	...	Face downwards
Hot	"	Left	"	"
"	"	Right	"	" upwards
Cold	"	Left	"	"

By Rotation around a Vertical Axis.

Rotation counter-clockwise	Head erect
" clockwise	" inverted

Note.—Inversion of the head was conveniently obtained by complete backward extension of the head over the end of a special rotating table, so that the head was about 10 in. from the axis of rotation.

TABLE IV.—HORIZONTAL NYSTAGMUS OBSERVABLE DURING ATTENTIVE DEVIATION AND FIXATION OF THE EYES TO THE LEFT.

By Thermal Methods.

Cold water irrigation	...	Left ear	...	Face downwards
Hot	"	Right	"	"
"	"	Left	"	" upwards
Cold	"	Right	"	"

By Rotation around a Vertical Axis.

Rotation clockwise	Head erect
" counter-clockwise	" inverted

TABLE V.—VERTICAL NYSTAGMUS OBSERVABLE DURING ATTENTIVE DEVIATION AND FIXATION OF THE EYES UPWARDS (IN RELATION TO THE ORBIT).

By Rotation Methods.

Rotation counter-clockwise	...	Right side of head downwards
" clockwise	...	Left " "

TABLE VI.—VERTICAL NYSTAGMUS OBSERVABLE DURING ATTENTIVE DEVIATION AND FIXATION OF THE EYES DOWNWARDS.

Rotation clockwise	...	Right side of head downwards
" counter-clockwise	...	Left " "

THE PHYSICAL EFFECTS OF HEAT.

I wish to associate myself with those who adopt the explanation of, I think, Breuer, concerning the nature of the thermal stimulus. It appears to me satisfactory, and is based upon the variations in density of watery fluids at different temperatures on the delicate tapering structure of the fibrillæ of the epithelial cells, on the ampullary crests of the semicircular canals and on the spatial arrangement of the ampullary cristæ of the three semicircular canals.

It is well known that as water cools the density of the cooler particles increases and they fall.¹ The more rapidly heat is abstracted the more rapidly do the particles fall. I see no reason to doubt that the endolymph possesses the same properties in these respects as other watery fluids, and that when the middle ear and outer wall of the labyrinth are cooled the perilymph and endolymph may be affected in accordance with the well-founded physical laws. If the temperature of the labyrinth is raised above the body temperature we can readily believe, for reasons which I shall give, that the phenomena observed are due to ascending convection currents in the endolymph. When the head is erect these descending or ascending currents would pass through the ampulla of the superior semicircular canal. When the face is directed upwards or downwards the currents would pass through the ampulla of the external semicircular canal. (The position of the ampulla of the posterior semicircular canal precludes satisfactory investigations by convection currents—a fact which supports Breuer's hypothesis.)

THE PHYSICAL EFFECTS OF ROTATION.

Inertia of the Particles of Endolymph and of the Fibrillæ.—At the beginning of all movements of the head the endolymph and the tapered ends of the fibrillæ will tend to remain stationary with respect to space. As soon as the inertia of the endolymph and of the tapered ends of the fibrillæ has been overcome by the frictional resistance² between the inner surface of the membranous labyrinth and the particles of endolymph in contact with it and with each other, the endolymph and tapered fibrillæ will acquire the same velocity through space as that at which the head is moving. When the head comes to rest the particles of endolymph will tend to continue their flow until the momentum they have acquired is overcome by the frictional resistance of the particles; the tapering fibrillæ will remain deflected until the forces causing deflection have been overcome by the elasticity of the fibrillæ.

¹ The law applies to ordinary atmospheric pressures, providing the temperature is higher than 4° C.

² The above is better stated in the terms of the physicist as follows: "When a cylindrical or, presumably, an ellipsoidal column of liquid is set moving in a tube, the velocity at different distances from the axis of the tube is well known to vary. The theory was originally given by Poiseuille (*"Mémoires des Savants étrangers,"* 1846). In a cylindrical tube the velocity falls off from axis to periphery according to the parabolic law, and in an ellipsoidal tube, when the ellipticity is not very marked, as in the case of the semicircular canals, this law would not be much departed from." (I am indebted to Dr. Frederick Womack, Lecturer on Physics to St. Bartholomew's Hospital, for this statement.)

COMPARISON OF THE HYPOTHESES OF CONVECTION CURRENTS AND
MOMENTUM CURRENTS.

We have seen reason to believe that convection currents may flow either from the utricle to the semicircular canals or from the semicircular canals to the utricle through the ampullæ, and by comparison of the forms of nystagmus induced by rotation we may infer that momentum currents may be set up in the endolymph in corresponding direction. The following tables indicate the direction of deflection of the three sets of fibrillæ which would be produced in accordance with the laws of convection considered in relation with a known anatomical structure.

Note.—The nature of the cupola of the fibrillæ has always appeared to me dubious, whether it is produced by deposits in the process of preparation of sections or not. I have excluded it in considering the supposed flexions of the fibrillæ. If the cupola represents a mass present during life it would aid in the mechanism of inertia and momentum.

*Reconsideration of the Stimulus in the Light of the preceding Physical Hypotheses. Further Details of Research.*TABLE VII.—DEFLECTION OF THE FIBRILLÆ OF THE SUPERIOR SEMICIRCULAR CANAL
ON ONE SIDE.

<i>By Thermal Method.</i>									
Direction of deflection of superior fibrillæ	Produced by			Position of head	Resulting type of nystagmus			Direction of maximum intensity	
From canal to utricle	...	Cold	...	Erect	...	Rotatory	...	Non-stimulated side	
" " "	...	Hot	...	Inverted	...	"	...	" "	
From utricle to canal	...	"	...	Erect	...	"	...	Stimulated side	
" " "	...	Cold	...	Inverted	...	"	...	" "	

Note.—No reactions are obtained where the method is applied to a defunct labyrinth.

TABLE VIII.—DEFLECTION OF THE FIBRILLÆ OF ONLY ONE SUPERIOR SEMICIRCULAR CANAL BY ROTATION WHEN ONLY THE RIGHT LABYRINTH IS FUNCTIONAL, THE LEFT BEING COMPLETELY DEFUNCT.

Direction of deflection of superior fibrillæ in right side			Direction of rotation		Position of head		Type of nystagmus	Direction of maximum intensity		
From canal to utricle			...	Clockwise	...	Face downwards	...	Rotatory	...	Left
"	"	"	...	Counter-clockwise	...	" upwards	...	"	...	"
"	utricle to canal		...	"	...	" downwards	...	"	...	Right
"	"	"	...	Clockwise	...	" upwards	...	"	...	"

TABLE IX.—DEFLECTION OF THE FIBRILLÆ OF ONLY ONE SUPERIOR SEMICIRCULAR CANAL BY ROTATION WHEN ONLY THE LEFT LABYRINTH IS FUNCTIONAL, THE RIGHT BEING COMPLETELY DEFUNCT.

Direction of deflection of superior fibrillæ in left side	Direction of rotation		Position of head		Type of nystagmus	Direction of maximum intensity	
From utricle to canal	...	Clockwise	...	Face downwards	...	Rotatory	... Left
" " "	...	Counter-clockwise	...	" upwards	...	"	... " "
" canal to utricle	...	"	...	" downwards	...	"	... Right
" " "	...	Clockwise	...	" upwards	...	"	... " "

Deflection of the Fibrillæ of both Superior Semicircular Canals simultaneously.—The nystagmus evoked by rotation clockwise and counter-clockwise with the face upwards and face downwards is precisely the same in type and directional characters when both labyrinths are intact as when only one is functional.

TABLE X.—DEFLECTION OF THE FIBRILLÆ OF THE EXTERNAL SEMICIRCULAR CANAL, EITHER SIDE SINGLY.

By Thermal Method.

Direction of deflection of external fibrillæ	Produced by	Position of head	Resulting type of nystagmus	Direction of maximum intensity
From canal to utricle ...	Cold water ...	Face downwards ...	Horizontal ...	Stimulated side
" " " ...	Hot " ...	" upwards ...	" ...	" " "
" utricle to canal ...	" " ...	" downwards ...	" ...	Non-stimulated side
" " " ...	Cold " ...	" upwards ...	" ...	" " "

TABLE XI.—DEFLECTION OF THE FIBRILLÆ OF ONLY ONE EXTERNAL SEMICIRCULAR CANAL BY ROTATION WHEN ONLY THE RIGHT LABYRINTH IS FUNCTIONAL, THE LEFT BEING COMPLETELY DEFUNCT.

Deflection of the external fibrillæ	Direction of rotation	Position of head	Type of nystagmus	Direction of maximum intensity
From canal to utricle ...	Counter-clockwise ...	Erect ...	Horizontal ...	To the right
" utricle to canal ...	Clockwise ...	" " ...	" " ...	" " left

Note.—The corresponding reactions are reversed when the rotation is made with the head inverted.

TABLE XII.—DEFLECTION OF THE FIBRILLÆ OF ONLY ONE EXTERNAL CANAL BY ROTATION WHEN ONLY THE LEFT LABYRINTH IS FUNCTIONAL, THE RIGHT BEING COMPLETELY DEFUNCT.

Deflection of the external fibrillæ	Direction of rotation	Position of head	Type of nystagmus	Direction of maximum intensity
From utricle to canal ...	Counter-clockwise ..	Erect ...	Horizontal ...	To the right
" canal to utricle ...	Clockwise ...	" " ...	" " ...	" " left

Note.—The corresponding reactions are reversed when the head is inverted.

Deflection of the Fibrillæ of both External Semicircular Canals simultaneously when both Labyrinths are Functional.—The nystagmus produced by rotation clockwise and counter-clockwise with the head erect or with the head inverted is precisely the same in type and directional characters when both labyrinths are intact as it is when only one labyrinth is functional.

TABLE XIII.—DEFLECTION OF THE FIBRILLÆ OF THE POSTERIOR SEMICIRCULAR CANAL, EITHER SIDE SINGLY, THE ONE BEING DEFUNCT, OR BOTH SIDES SIMULTANEOUSLY, BOTH LABYRINTHS BEING FUNCTIONAL.

Direction of deflection of the inferior fibrillæ, i.e., of posterior canal	Direction of rotation	Position of head	Type of Nystagmus	Direction of maximum intensity
From utricle to canal ...	Clockwise ...	Right side downwards	Vertical ...	Downwards
" " " ...	Counter-clockwise ...	Left side downwards	" ...	"
" canal to utricle ...	Clockwise ...	" " "	" ...	Upwards
" " " ...	Counter-clockwise ...	Right side downwards	" ...	"

The Posterior Semicircular Canals.—The position of these canals precludes the complete investigation which is possible in the case of the horizontal and superior canals. Although set in a plane which forms an angle of about 30° with the sagittal plane the effects of rotation in the sagittal plane are manifest after one posterior canal has been destroyed. In some cases of unilateral ablation one has attempted by a series of trials to adjust the head on the turntable by sand-bags so that the plane of rotation shall be at right angles to the normal plane of the remaining posterior canal. This position brings the superior semicircular canal on the same side into the plane of rotation, and the resulting nystagmus is rotatory, not vertical.

THEORETICAL DATA OF ROTATION.

Greater effort may be required to provoke nystagmus by rotation in one direction than by rotation in the opposite direction, and some observers hold that each ampullary nerve has a "principal" and a "subordinate" function. (Lee, loc. cit.; cf., Bárány, loc. cit.)

The following reasons appear to me to be opposed to this view; there is good reason to think that greater effort is required to set up momentum currents from the utricle through the ampulla to the canal than from the canal through the ampulla to the utricle. After the set of semicircular canals on one side has been removed, it will be found that nystagmus can be produced by less number of revolutions in one direction than are necessary to provoke nystagmus by rotation in the opposite direction. (The facts can be better demonstrated in cases in which there is no spontaneous nystagmus.)

Viewing the matter physically, in order to estimate the momentum set up by rotation we must pay regard to the angular velocity, tangential speed and duration of rotation. The angular velocity is determined by computing the time of one complete revolution of 360° , and stated as so many degrees per second. If we adopt a uniform angular velocity of, say, 360° in five seconds, the approximate tangential speed varies directly with the distance between the semicircular canals and the axis of rotation; this distance can be obtained by measuring the radius of the curve of revolution described by the external auditory meatus, which will give the approximate relative position of each set of the semicircular canals.

It will be obvious that when the patient reclines on the turntable with the head half a metre from the axis of rotation, and the table be turned at the uniform angular velocity of 360° per five seconds, the

tangential speed of the head, and therefore of the labyrinth and of the endolymph, will be greater than when the head is only a quarter of a metre from the axis of rotation. Similarly, if the patient be seated over the axis of rotation with the labyrinths equidistant from the axis of rotation, the tangential speed will be equal in the two labyrinths. But if the patient be seated a little to one side of, and not quite accurately over the axis of rotation, so that the labyrinths are not equidistant from that axis, then greater tangential speed will be set up in the labyrinth which is at the greater distance from the axis of rotation. Now, when mass and angular velocity are constant, the strength of the stimulus—which depends upon the arrest of momentum—varies directly with the tangential speed. Owing to the inequality of the sectional areas of semicircular canal and ampulla it will take a different time to establish uniformity of movement throughout the whole body of the endolymph when the head is rotated uniformly in one direction from that which it will take if rotated uniformly in the opposite direction, the current in the one case being from narrow to broad channel and in the other from broad to narrow, the sectional areas of the two portions being as 25 to 1.¹

SECTIONAL AREAS OF MEMBRANOUS LABYRINTHS.

Seeing that these canals are only slightly ellipsoidal, and nearly circular in section, we may estimate the relations of their sectional areas by a comparison of the squares of their radii. We shall find that the relation of the sectional areas of the membranous canal and the bony canal can be expressed approximately by 1 to 6·25. And the relation of the sectional areas of the membranous canal and the membranous ampulla is computed as being between 1 to 25 and 1 to 45. (Note the membranous ampulla nearly fills the bony ampulla, as shown by histological specimens.)

Transverse diameter of membranous and bony ampulla	...	1·50 mm. to 2 mm.
" " bony semicircular canal	...	0·75 "
" " membranous semicircular canal	...	0·30 "

THE THEORY OF THE STIMULUS PRODUCING LABYRINTHINE NYSTAGMUS.

If we recall the normal circumstances of stimulation of the ampullary nerves by ordinary head-movements, and regard the stimulus as being

¹ I have had the advantage of conferring with Dr. Frederick Womack, Lecturer in Physics at St. Bartholomew's Hospital, in expressing these conclusions.

produced by flexion of the fibrillæ due to inertia of the free ends of the fibrillæ and of the surrounding endolymph, we must regard any stimulation by moving currents as abnormally intense. To produce such currents by rotation greater angular velocity or tangential speed or duration of rotation is required to overcome the frictional resistance of the endolymph in one direction than in the other. But in normal conditions of stimulation by inertia, no currents are set up and the stimulus will be as effective when the head is moved in one direction as when it is moved in the other. This view is not in accord with that of others who hold that the ampullary nerves possess a "principal" and a "subordinate" function, nor does it agree with those who assume that the stimulus set up by *normal* head-movements in one direction acts more strongly on any one particular crista than the stimulus produced by normal head-movements at the same angular velocity in the opposite direction.

Although I believe there is a double function I do not believe that one is more potent than the other, for, as I shall show, the theory of principal and subordinate functions is opposed to the facts of binocular compensatory movement being preserved, after removal of one set of semicircular canals on one side, however well it may explain the movements of deviation which have been observed after the loss of one labyrinth in fish (*see* Lee, loc. cit.).

I have made all the foregoing observations on numerous occasions and in a large number of individuals of various ages, and have no hesitation in accepting the explanations given of the stimulation which sets going the mechanism of nystagmus.

NYSTAGMUS ASSOCIATED WITH LABYRINTHINE FISTULA (EXTRA-AMPULLARY LESIONS).

Careful consideration of the nature of the stimulus of labyrinthine nystagmus can be applied to explain nystagmus associated with fistulous erosions of the osseous labyrinth, *when the membranous ampullary apparatus is still intact*. For instance, in a case of fistula of the external semicircular canal which was covered with dry cholesteatomatous material, there was no spontaneous nystagmus. Normal nystagmus reactions were produced by rotation tests and by caloric tests on both sides. Horizontal nystagmus was easily provoked by sudden slight compression of the meatus on the affected side. Rarefaction of the meatus did not produce nystagmus. Pressure on the

fistula with a probe armed with a small pledget of wool produced the same type of nystagmus as produced by meatal compression. The rapid movement was towards the side affected. When the pressure was increased, horizontal and rotatory nystagmus was induced.

The nystagmus in this case appeared to be due to pressure transmitted to the membranous external semicircular canal, posterior to its ampulla, which would cause deflection of the fibrillæ of the external crista towards the utricle and horizontal nystagmus to the affected side. (See Table X.)

In another case of fistula of the external semicircular canal, the antrum was filled with soft vascular granulation tissue, which covered the fistula. In this case there was spontaneous horizontal nystagmus during attentive fixation of the eyes towards the affected side. The intensity of the nystagmus was increased by sudden meatal compression and the patient reeled and nearly fell down. The spontaneous nystagmus in this case may have been produced by the transmission of pulsation from the mass of granulations in the antrum over the fistula, through the endolymph of the external canal to the crista of the external ampulla, with resulting deflection of the fibrillæ forwards towards the utricle.

In cases of fistula of the outer vestibular wall, associated with normally reacting ampullary systems (as ascertained by the nystagmus resulting from caloric tests) spontaneous nystagmus appears to be connected with the presence of vascular granulations in the tympanum.

In a case of fistula of the fenestra ovalis which I described elsewhere¹ there was no spontaneous nystagmus, but vertigo was induced by meatal compression. In cases of vestibular fistula in a normally reacting labyrinth the spontaneous nystagmus, when present, was rotatory in type, or combined rotatory and horizontal; the explanation one offers is that transmission of pulsation takes place from granulations to the endolymph from the utricle towards the superior and external canals. (See Tables VII and X.) Whether these suppositions will explain all apparently similar cases remains to be seen.

THE RELATIONS OF THE SEMICIRCULAR CANALS TO NORMAL OCULAR FIXATION.

The inferences which I have drawn from a study of these data have led me to regard the ampullary apparatus as the receptive organ of stimuli set up by deflection of the fibrillæ; that under ordinary conditions the deflection is produced by movements of the

¹ *Lancet*, 1907, ii, p. 1677.

head; that the force causing the deflection is due to inertia of the fine ends of the fibrillæ; that the impulses are conveyed by the ampullary nerves to the brain; that these afferent impulses affect among other parts the oculo-motor apparatus and cause a movement of the eyeball; that this movement of the eyeball aids attentive fixation of the visual axes upon an object during movements of the head; that the direction of movement of the eyeball depends upon the source of the stimulus; when the stimulus originates in the ampulla of the external semicircular canal the direction of ocular movement is horizontal; when the stimulus arises in the ampulla of the superior canal the movement of the eyeball is rotatory, and when the stimulus originates in the inferior ampulla of the posterior semicircular canal the movement of the eyeball is vertical. Moreover, when stimuli arise simultaneously in the external and superior semicircular ampullæ the resultant eye-movement is composed of horizontal and rotatory elements; that when the stimuli originate simultaneously in the superior and inferior ampullæ the resultant movement is composed of rotatory and vertical elements; and that when the stimuli arise simultaneously in the external and inferior ampullæ the resultant eye-movement is oblique. And similarly when stimuli originate in all three canals simultaneously the resultant eye-movement is composed of horizontal, vertical and rotatory elements. The direction of eye-movement in any particular plane depends upon the direction of deflection of the fibrillæ in the ampulla in which the stimulus originates. The rapidity and extent of movement of the eyeball varies directly with the intensity and duration of the stimulus. The intensity and duration of the stimulus will vary directly with the rapidity and period of movement of the head.

Thus we infer that there is a perfect correspondence between the direction, rapidity and extent of movement of the eyeball and the direction and period of movement of the head. I think it will be admitted that by this correspondence between head- and eye-movements spatial position of the visual axes is controlled and we are enabled to preserve accurate attentive visual fixation notwithstanding continual movements of the head.

OBSERVATIONS ON OCULAR FIXATION DURING IMMOBILIZATION OF THE HEAD.

I shall attempt to show that the primary movement of labyrinthine nystagmus is an exaggeration of this reflex movement which normally controls fixation. But we must first consider what are the normal

movements of the eyeball which maintain fixation on moving objects during immobilization of the head. Last year I presented a paper¹ to the Anatomical Society of Great Britain and Ireland, from which I shall quote the following paragraph:—

Determination of the functions of the normal labyrinth in relation to the control of normal ocular fixation. . . . Let us first consider what form of eye-movements take place when the head is kept erect and motionless, while the eyes are directed upon a small moving object, such as the end of a pencil, held by an observer about 18 in. distant from the eyes. The individual under observation is directed to follow every movement. . . . If the pencil is moved from one side to the other within the visual field at a rate not exceeding about 45° per second the eyeballs appear to move regularly and smoothly in adjusting fixation, and the image is kept focussed in the line of most distinct vision. If the pencil or other object be moved more rapidly across the visual field the attempt to preserve fixation can be seen to fail, for the eyeballs move in obvious jerks in the effort to regain fixation.²

Movements of the eyeball made in the effort to regain fixation on a moving object are called by Dr. Dodge "the movements of pursuit"—a name which commends itself. His experimental conditions differ from my own, for the photographic method does not lend itself to wide ranges of movement such as I have described, and Dr. Dodge does not demonstrate with the camera what is obvious to the naked eye—namely, that the eye moves in a successive series of short rapid jerks for movements of pursuit upon objects moving at a greater angular velocity than about 45° per second. In explaining what appears to me to be the part played by the simple movement of pursuit in the mechanism of nystagmus, I shall refer to this phenomenon as the "simple reflex action of attentive pursuit."

OCULAR FIXATION DURING HEAD-MOVEMENTS.

Let us now consider what happens when the object is at rest and the head is moved while ocular fixation is maintained. This can be ascertained in a normal person who is directed to hold the head erect and to fix the eyes upon an object about eighteen inches away. The head is then turned slowly from side to side as in expressing a negative sign—

¹ Withdrawn from publication by Mr. Scott owing to issue of the present paper.

² I communicated this observation to Dr. Henry Head, who told me of a paper about to appear in the November number of *Brain*, which was in the press, in which the authors described movements of the eyeball during immobilization of the head, illustrated by records obtained by photo-chronographic methods. See "An Experimental Study of the Ocular Reactions of the Insane from Photographic Records," by Dr. Allen Ross Diefendorf and Dr. Raymond Dodge, *Brain*, Lond., 1908, xxxi, p. 451.

i.e., the head is rotated from side to side around a vertical axis, through about 90° . When the head moves at the velocity of 45° per second perfect fixation is preserved without effort; and it will be found possible to increase the velocity of movement of the head from side to side up to 90° per second and even 180° per second and still maintain ocular fixation, unaccompanied by any jerking movement of the eyes.

Perhaps it is not necessary to say that those movements depend upon normal visual powers. What has been said regarding ocular fixation on objects moving horizontally can be applied to fixation on objects moving vertically. Similarly the observations on fixation during lateral movements of the head may be applied to fixation during nodding movements.

Moreover, it is known to physiologists and ophthalmologists that, when the eyes are fixed upon an object at rest and the head is flexed slowly to one side, the eyeball is maintained, within certain limits, in the original position in space by rotating in a direction opposite to that of the head. In other words, the inclination of the horizontal meridian of the eyeball, which lateral inclination of the head would produce, is corrected by a compensating rotatory movement of the eyeball about its antero-posterior axis in the opposite direction. This can readily be demonstrated by watching two spots on the iris taken in the same horizontal plane, one on each side of the pupil. Upon slight lateral inclination of the head the two spots remain at the same level. As the lateral flexion of the head increases, the limit of ocular adjustability to maintain constant fixation is soon reached, and the eyeball rotates with a rapid jerk in the direction of inclination.

SYNTHESIS OF LABYRINTHINE NYSTAGMUS. THEORY OF ITS MECHANISM.

We can now endeavour to show that the simple reflex action of attentive pursuit is the secondary of the two movements which constitute labyrinthine nystagmus. The primary movement of nystagmus is an exaggeration of the normal reflex movement of labyrinthine control, and is produced in response to a stimulus so intense that the eyeballs are deviated to their extreme limits. When the stimulus is a simple one, originating in one ampulla only, the eyeballs are deviated reflexly in one direction, in which position there is no nystagmus. If an attempt be now made to turn the eyes attentively in the opposite direction away from the direction of deviation, the reflex movement of deviation overcomes the movement of attention (vicious control), and gives rise to a

succession of fleeting images sweeping across the retina, and an impression of objects moving rapidly towards the other side. The simple reflex action of attentive pursuit now comes into play, and the eyes are rapidly jerked in pursuit of the apparently moving object, which is momentarily fixed; immediately labyrinthine reflex deviation gains the upper hand, images once more flee across the retina, causing once more the simple reflex action of attentive pursuit. And so on until the labyrinthine stimulus and the reflex deviation ceases, when the nystagmus comes to an end. But this does not explain all. Associated with these alternating reflex acts is a whole series of psychical impressions; and, moreover, they are in turn accompanied by impressions connected with apparently the whole musculature of the body.

THE CRUM-BROWN THEORY OF PAIRED CANALS.

Professor Crum-Brown's original description of the impressions arising from the paired canals led him to conclude that "one canal can . . . be affected by and transmit the sensation of rotation *about one axis in one direction only*, and for complete perception of rotation in any direction about any axis six semicircular canals are required, in three pairs, each pair having its two canals parallel (or in the same plane) with their ampullæ turned opposite ways."¹ While agreeing with the anatomical facts as stated by Crum-Brown it seems to me that a modification of his views is necessary to explain the functions of the canals. In the first place, one set of semicircular canals on one side is capable of performing the functions of two sets of canals with very little loss of precision, as shown by the results of destruction by disease or operation of one labyrinth. In the second place, the results of rotation in the plane of, and in planes adjacent to, that of either remaining posterior semicircular canal cause vertical movements of the eyes, whether the direction of movement of the head is such that the ampulla moves first or last. Also when the rotation is made in the plane of the remaining superior semicircular canal the ocular movements are purely rotatory and about the anterior posterior axis of the eyeball. When both sets of semicircular canals are intact, movement of the head by rotation in the plane of the ^{right} superior semicircular canal and in the plane of the ^{left} posterior canal simultaneously causes combined vertical and rotatory

¹ *Journ. of Anat. and Phys.*, 1874, viii, p. 330; also quoted by Ferrier: "The Functions of the Brain," 1876, p. 59.

movements of the eyeball, compensatory to the direction of movement of the head, for this direction of movement of the head in the plane of one superior semicircular canal and of the opposite posterior semicircular canal can be resolved into component sagittal and lateral vertical planes. According to the Crum-Brown theory one only of the paired canals is stimulated. It seems to me that each paired canal is stimulated during movement of the head in planes coinciding or nearly coinciding with the plane of the canal. That when one set of these canals on one side has been completely destroyed the other set is capable of controlling compensatory movements of the eye, although there will not be quite such precise control in certain limited direction of head-movement, viz., in mathematically exact planes of either the remaining superior or posterior canals. Thus instead of combined vertical and rotatory movements of the eye during attentive fixation resulting from head-movements in the exact plane of the posterior canal, or at right angles to the crista of the inferior ampulla, only vertical eye-movements occur; and during head-movements in the exact plane of the remaining superior canal or at right angles to the crista of the superior ampulla only rotatory eye-movements are produced. During ordinary circumstances the individual may lose little or nothing as regards compensatory power of eye- and head-movement after one set of semicircular canals has been removed. Under certain circumstances the loss of one set of semicircular canals interferes with compensatory eye-movements; this occurs when unilateral ablation of one labyrinth is associated with persistent spontaneous nystagmus and giddiness.

SPONTANEOUS NYSTAGMUS FOLLOWING COMPLETE DESTRUCTION OF ONE LABYRINTH (ABLATION NYSTAGMUS).

After recent destruction of one labyrinth in the adult human subject spontaneous nystagmus is generally present at some time or other, and presents the usual characteristics of labyrinthine nystagmus, provided one labyrinth remains functional. When present the nystagmus varies in intensity from time to time. It may either be rotatory, or combined rotatory and horizontal, or combined rotatory, horizontal and vertical [rotatory and oblique]. I have never seen pure vertical nystagmus as an isolated type of nystagmus in cases of unilateral ablation, nor pure horizontal nystagmus alone; the rotatory element has been invariably present, and in the slighter cases rotatory nystagmus occurs alone. In some persons giddiness and nystagmus are very severe, and occur upon

attentive deviation of the eyes to either side. In other cases giddiness and nystagmus are well marked when the eyes are attentively turned towards the normal side. Lastly, there may be no spontaneous nystagmus after unilateral ablation, even though nystagmus reactions can be obtained by rotation and by caloric stimulation of the intact labyrinth.

THE EFFECT OF REMOVAL OF ALL SIX SEMICIRCULAR CANALS
(BILATERAL ABLATION).

I have only seen one case of bilateral ablation of the labyrinth—Mr. Lake's classical case of extirpation of both internal ears.¹ Through his courtesy I have been enabled to examine this patient on the rotation table. In spite of twenty continued revolutions around a vertical axis with the head placed 20 in. from the axis of rotation (face upwards) at the angular velocity of 180° per second (which represents a relatively enormous tangential speed) no nystagmus developed, no giddiness was felt; the patient got up immediately, unaided, without the least sign of discomfort of any kind. Her reactions of simple attentive pursuit, as indicated by eye-movements in fixing moving objects during immobilization of the head were obviously more acute than in a person with normal labyrinths. This was several years after the removal of the labyrinths. Although fixation of pursuit during head-movements was astonishingly good, it was imperfect.

A NEW HYPOTHESIS TO EXPLAIN UNILATERAL ABLATION NYSTAGMUS.

To uphold a new hypothesis the following features of spontaneous unilateral ablation nystagmus appear to me to be the most important: That it is absent after the removal of the labyrinth in young subjects; that when present after recent ablation in young adults with normal vascular systems it is generally slight; that in my experience, when carefully looked for, it has been found to accompany recent ablation in persons after about 35 years of age.

When very severe the nystagmus is rotatory in type to one side and oblique or horizontal to the other. The rotatory movement has always been a fine movement clockwise, best seen on attentive deviation of the eyes to the left, or counter-clockwise on deviation to the right of the binocular field. The rotatory nystagmus is best observed upon attentive deviation towards the normal side. The horizontal or oblique nystagmus

¹ *Proc. Roy. Soc. Med.*, 1908, 1 (Otol. Sec.), p. 150.

has been observed when the eyes are attentively deviated towards the side of the removed labyrinth. The horizontal and oblique movements have a wider range than the movement of rotatory nystagmus. I think we may regard the oblique movement, which is directed obliquely downwards and towards the side of attentive deviation, as being composed of horizontal and vertical elements, the latter being directed vertically downwards; that is to say, we have the three elementary types of labyrinthine nystagmus which correspond to those produced by stimulation of the three ampullæ. In the course of time this spontaneous nystagmus becomes less marked. The first element to disappear is the vertical element, so that we witness a stage in which there is rotatory nystagmus to one side and pure horizontal nystagmus to the other.

At a later period, sometimes several weeks, the latter disappears, and rotatory nystagmus remains alone. This is the type of nystagmus which is most commonly seen; it is sometimes so slight as to be overlooked. It occurs on attentive deviation only to one side. The asymmetry aids its recognition. It may disappear and occasionally recur during intervals extending over a period of several years—*e.g.*, four years. In some cases, in which it seems to have disappeared, it may be made to reappear by over-exertion, and by certain other circumstances.

Analysis of the above-described Triple Type of Spontaneous Nystagmus.—If we analyse the above composite type of nystagmus we shall conclude that the nystagmic movements are the same as those which would be produced by stimuli which cause deflection of the fibrillæ of the ampullary cristæ in the normal labyrinth from the utricle towards each of the three semicircular canals simultaneously; that is to say, the fibrillæ in the superior ampulla would be deflected upwards, and the fibrillæ of the external and inferior ampullæ would be deflected backwards, and we should set out to seek an agent to the influence of which such deflections may be attributed. (*See Tables I to XII.*)

The Influence of Immobilizing the Head.—The effect of immobilization was tried in several patients with well-marked ablation nystagmus, but in no instance was the nystagmus arrested. The patients were kept at rest on a wooden couch in a room with concrete floor for periods of fifteen to thirty minutes, with the head immobilized between sandbags.

The Influence of Bodily Rest without Immobilization of the Head.—In some cases in which there was slight unilateral nystagmus bodily rest was followed by disappearance of the nystagmus, even when the head was not immobilized. In these cases there had been some hurried

movements or apparent mental excitement on the part of the patient before the effect of rest was noted.

The Influence of Muscular Exertion.—In certain cases of recent unilateral ablation, in which there was no spontaneous nystagmus at the time of examination, I found it possible to provoke nystagmus which was rotatory in type and which appeared on attentive deviation of the eyes to the normal side by exercises, which caused the heart-beat to increase in frequency from about eighty to hundred or more beats per minute. After a few minutes' rest, during which time the heart-beats returned to the normal, the nystagmus disappeared. In other cases in which there was slight spontaneous rotatory nystagmus to the sound side, muscular exercises which caused increased force of the pulse were followed by additional horizontal nystagmus to the ablated side. In another case in which there was spontaneous rotatory nystagmus to the sound side and horizontal nystagmus to the ablated side, it was noticed that muscular exertion was followed by conversion of pure horizontal nystagmus to the ablated side, into oblique nystagmus downwards and to the ablated side.

I cannot conceive that such changes in the type of nystagmus as these which followed physical exercises of the arms can be attributed to changes in the nerve-fibres of the labyrinth which had been destroyed, and although I have not yet studied Wallerian degeneration in the auditory nerve, I feel justified in assuming that the effect of exertion must be sought for in the *normal labyrinth*.

The Influence of Carotid Pulsation.—I would remind you that the internal carotid artery runs through the carotid canal of the petrous bone below and in front of the cochlea, where it makes a somewhat abrupt curve forwards and upwards. It needs little imagination to realize that if the heart-beat is sufficiently strong, or if there is sufficient deficiency of elasticity in the walls of the artery, some of the force of the heart-beat may be imparted to the walls of the carotid canal and may possibly reach the labyrinth in the form of an impaction wave.

The Effect of Compression of the Carotid Artery on the Normal Side.—In order to ascertain whether the increased nystagmus could be attributed to the direct influence of the carotid pulsation, the carotid arteries were compressed first on one side and afterwards on the other. When the common carotid artery was compressed against the cervical vertebræ on the side of the labyrinth which had been destroyed, no influence on the nystagmus was observable in eight successive patients who had spontaneous ablation nystagmus. But when the common

carotid artery was compressed on the side of the normal labyrinth, the nystagmus was completely arrested in each case. As long as the compression was continued nystagmus was unobservable, and when the artery was released the spontaneous nystagmus returned. Moreover, patients with severe spontaneous ablation nystagmus accompanied by giddiness tell me they completely lose all sensation of giddiness during carotid compression.

The Hypothesis of Impaction Waves causing Spontaneous Ablation Nystagmus.—If such a wave as that which I have suggested exists, it should be imparted to the petrous bone where the internal carotid artery makes its forward curve. Such a wave would traverse the narrow capsule of the labyrinth which intervenes between the labyrinthine fluid and the walls of the carotid artery. The direction of transmission of such a wave would be chiefly upwards and backwards, from the convex arch of the carotid canal. In this direction the wave would traverse the vestibule and the semicircular canals. We should suppose the most constant waves would travel across the utricle towards the ampulla of the superior canal. We may well conceive such a wave would have greatest influence on the marginal deflection of the tapered extremities of the fibrillæ upwards. We can conceive that if the impaction wave be sufficiently strong it would affect the ampulla of the external semicircular canal, and waves reaching the inferior ampulla would only do so when the force of impaction is usually vigorous. Now if such impaction waves do actually exist, and if they acted in the way I have suggested, the fibrillæ of all three ampulla would be deflected from the utricle towards the canals simultaneously, and the type of nystagmus which these deflections would produce should be rotatory towards the stimulated side, and horizontal towards the non-stimulated side, and vertical downwards (that is, oblique downwards and towards the non-stimulated side). And this is the type of nystagmus which I have found to last for several weeks after one labyrinth has been extirpated in adults who have unusually forcible pulsation of the carotid artery.

Application of the Hypothesis to a Case of long-standing Unilateral Defunct Labyrinth.—Assuming such impaction waves do exist, it is interesting to consider the possible ultimate effect on the eye-movements of forcible carotid pulsation. A case came under my observation. A man aged about 60 had had suppurative disease of the left middle ear in early life (fifty years previously), which had left him totally deaf to bone conduction on that side. The inner tympanic wall could be distinctly seen. The effect of heat and cold to the left tympanum produced no

nystagmus and no giddiness whatever; the other ear was normal as revealed by hearing tests combined with the easily obtained normal reactions of nystagmus in response to hot and cold irrigation of the right ear. Thus it seemed that the internal ear on the left side was defunct, and that the right internal ear was normal. There was no spontaneous nystagmus.

COMPRESSION OF THE CAROTID IN THE NORMAL SIDE.

The absence of nystagmus in this case can be explained either by the absence of impaction waves or by the acquired disregard of impaction waves. If we may assume for a moment that this patient's right labyrinth had for many years been subjected to the shocks transmitted to it from the internal carotid artery, the fibrillæ of the superior semicircular canal would be constantly deflected upwards, causing a stimulus which the patient had learnt to disregard. If this be the case, and the impaction waves be suddenly cut off by compression of the right common carotid artery, the effect on the labyrinth in removing the stimulus should be the same as that induced by a stimulus of the opposite kind—namely, *relatively* downward deflection of the superior fibrillæ—and we should expect rotatory nystagmus clockwise in direction to appear during carotid compression upon deviation of the eyes to the left—that is, away from the “stimulated” side. (Table VII.) Now it is a most remarkable fact that this was the actual effect of compressing the right carotid artery in this man. There was no giddiness and no spontaneous nystagmus on attentive deviation of the eyes in any direction before the artery was compressed, and directly the right artery was compressed so as to cause disappearance of the pulsation in the superficial temporal artery on that side, the patient became severely giddy, rocked about in his chair, and rotatory nystagmus, clockwise in direction, appeared upon attentive deviation of the eyes to the left side. These observations were repeated and the results were constant, and were witnessed by others present at the time. Compression of the left carotid produced no symptoms whatever.

CONCLUSION.

It is not wise to make any premature generalizations, and I am content to record the facts as I observed them that they may be tested independently by others. If my observations can be substantiated by competent observers in similar cases of unilateral ablation of the

labyrinth, I think some new data with which to reconsider the problem of vertigo and the mechanism of nystagmus may be obtained.

And I think it may possibly be found that various forms of nystagmus hitherto unassociated with labyrinthine stimulation bear such a striking resemblance to labyrinthine nystagmus that it is difficult to dissociate the causes of these forms of nystagmus from those which are acknowledged to be connected with stimulation of the semicircular canals.

And lastly, I bring these observations forward with the hope that any discussion which they may arouse will lead to a conception of more definite views of the origin of vertigo in cases of atheroma; the causes of giddiness produced by digital compression¹ and like conditions, and I hope that my remarks will not be considered useless in considering the more precise indications and contra-indications for the operations which have been designed for the relief of vertigo.

Note.—I desire to express my great appreciation of the generosity of my colleagues, Mr. Cumberbatch and Mr. West, for the opportunities they have afforded me of making observations on many of their patients.

¹ Since this was written I have turned to Jacobson's "The Operations of Surgery," 4th ed., 1902, i, p. 585. In speaking of the treatment of carotid aneurysm he quotes from the "Encyclopedia of Surgery": "If pain, vertigo, sickness, &c., prevent a fair trial of digital pressure . . ." Erichsen's "Science and Art of Surgery," 10th ed., 1895, ii, p. 176: "Cerebral symptoms" arise in 25 per cent. of cases of ligature of common carotid artery; "twitching and giddiness" are recognized among these "cerebral" symptoms.

DISCUSSION.

The PRESIDENT (Dr. McBride) expressed the indebtedness of the Section to Mr. Scott for his able communication. It was a subject which was difficult to discuss, but one well worthy of debate. He had himself read the paper twice, but felt he wanted to peruse it a third time before expressing very definite opinions.

Sir VICTOR HORSLEY, F.R.S., said he had never enjoyed the hospitality of the Section so much as on the present occasion. From the abstract of the paper which had been kindly sent him, it appeared to be the paper of its year. Members could not be sufficiently grateful to Mr. Scott for the enormous amount of work he had done on the subject, and now he had put forward clinically the facts differentiating the central and peripheral mechanisms. He had not only grouped the facts in a clear way, but had suggested clinically a complete explanation of the very difficult subject of compensation with peripheral disease of the eighth nerve. Having expressed gratitude, he was going now to ask for more. Because, if one approached the question of the effects on the peripheral mechanism of stimulation, by Bárány's or other methods, one was at once struck, as Mr. Scott had detailed so completely, with the prominence of the nystagmus. That was, however, not the only motor effect which should be studied in that connexion. The other motor phenomenon which was a striking feature in the peripheral stimulation of the vestibular nerve was the attitude of the head. He referred to the sinking of the ear to the homolateral side in a lesion unbalancing the reflex arcs, and that was of as much importance as the nystagmus. One of the statements which interested him most was that the removal of the labyrinth in the child was not essentially followed by nystagmus. He would suggest that that was because a child was nearer to the monkey than was the adult. During the last four or five years he had done a number of experiments on monkeys, on both the peripheral and central nervous mechanism, and it was remarkable that when one removed the labyrinth in a monkey, the head sank at once, so that the line from ear to ear was almost vertical. Yet when the monkey was nursed and its attention taken, and it was quite quiet, it was very difficult to see nystagmus after the first twenty-four hours. In that respect the monkey and the child were on the same platform. When one compared the results of direct electrical stimulation on the peripheral mechanism with that of the central mechanism, there was at once a difference, recognition of which would be diagnostically useful in the future; in that from the central mechanism one tended to get a nystagmus which was most pronounced in the ipsilateral eye, whereas in the peripheral stimulation both eyes moved to the same degree. He gathered that that was the experience of Bárány. It seemed to be the case also in cerebellar tumour. Mr. Scott had also furnished a very valuable aid in clinical diagnosis. As Mr. Scott had had an opportunity of investigating those cases of loss of labyrinth on one side, he (Sir Victor) asked

whether, when the patient was in a condition of stimulation and the nystagmus was in full swing as the result of stimulation of the opposite side, there was any hypertonia of muscles on either side of the body. There ought to be, and the point could be ascertained at the time of investigation. That brought him to the question of compensation. He had not seen Mr. Lake's classical case, but that case, and Mr. Scott's demonstration of the effect of placing the patient without a labyrinth on a turntable, interested him very much. When the right labyrinth was removed in a monkey, the head took up the forced posture, and remained in that condition for several weeks, after which time it gradually diminished. On removing the left labyrinth, the head at once became straight. This fact of compensation quite confirmed Mr. Scott's observation concerning the influence of the carotid pulse, though he was aware that was not the universal experience. Another point in regard to compensation for which they were grateful to Mr. Scott was his frank expression of disbelief in the so-called continual stimulation from the diseased side. Nothing like that was seen in normal physiology; one could not keep on stimulating a nerve in that way. If there was dissociation, and as a result the head was in an abnormal posture, or the eyes moving in a nystagmoid manner, it was because the disease had destroyed a portion of some symmetrically arranged system; it was not a question of prolonged stimulation from the seat of disease. He was very grateful for the opportunity of hearing such a valuable communication.

Mr. WAGGETT said he thought no greater compliment could be paid to the paper than the demand for more which came from Sir Victor Horsley. He wished to mention an interesting instance of hypertonus on the opposite side to that of the external semicircular canal which was stimulated by a movement towards its own side. He referred to the mistakes which were made when driving at golf. A good golfer kept his head still when driving, but the imperfect player moved his head suddenly to the left when giving the down swing, thus stimulating the crista of the left external canal. It was obvious that a stream passing from the narrow left semicircular canal stimulated the left crista, whereas the corresponding stream from the right ampulla into the right canal had no particular effect. The speaker believed that this point was not novel. The unsteady golfer, by stimulating the left external crista, induced hypertonus of the muscles on the right side; the right shoulder was thus drawn up to the head, and the arm was "pulled in," causing a "slice"; at the same time and for a similar reason the right knee was suddenly bent or "ducked," with the result that the head of the club struck the ground some inches behind the ball.

Dr. ALBERT GRAY said that in referring to labyrinthine stimulation it was a common error to speak of velocity, instead of acceleration. A constant velocity in a straight line or in a circle did not cause stimulation of the labyrinth, as was illustrated if one woke up after going to sleep in the train, the train being still in motion; by shutting the eyes again it was easy to imagine oneself going in the opposite direction. In a uniform movement all the constituent particles and parts remained in the same relation to each other. In reference to Mr.

Scott's experiments concerning particles suspended in fluid, it must be remembered that the direction which those particles would take depended on their specific gravity relative to that of the fluid. If the particles were lighter than the fluid they would advance in front of it, and if heavier they would lag behind. Nystagmus he regarded as a matter of evolution. The movements of the head, as mentioned by Sir Victor Horsley, he regarded as very important. The matter was well illustrated in the labyrinth of the porpoise, where the canals and nerves were very small, no doubt due to the inability of the animal to move its head on its trunk. Though this condition was present also in the sea-cow, it was less marked. With regard to the reason of the nystagmus after unilateral ablation in people after the age of 35, due possibly to arterial changes, he ventured to suggest that there might be another factor. At the prime of life retrograde changes began to take place in the neurons. Might not that explain nystagmus in these cases being more noticeable after 35 years of age? The paper was one of the most interesting he had listened to.

Dr. DAN MCKENZIE first of all expressed his gratitude to Mr. Scott for the opportunity he had afforded the Section of becoming acquainted with the latest work on the labyrinth. The speaker went on to say that in his historical survey of the subject Mr. Scott had omitted to mention the name of Ewald, although it was upon that observer's experiments that all the existing theories of labyrinth nystagmus had been based. Mr. Scott, in his discussion of the causation of rotation nystagmus, quoted the results obtained by the thermal tests in support of the inertia (or momentum) theory; on the other hand, the speaker was acquainted with certain writings where the process was reversed and where the momentum theory of rotation nystagmus was summoned to the defence of the convection theory of thermal nystagmus. But a mutual support of each other by these respective theories was unnecessary, since both were ultimately founded upon the results of Ewald's experiments. Referring to the explanations which had been given of rotation nystagmus, Dr. McKenzie pointed out that serious objections could be levelled against the theory that the nystagmus was wholly caused by currents of endolymph. These objections might be divided into two groups. In the first group came the physical objections, some of which were as follows: Mr. Scott had told them that the diameter of the membranous canal proper was only $\frac{1}{3}$ mm., or, as Bárány put it, equal to that of a household pin. In other words, its dimensions approached those of a capillary tube, and without doubt the frictional resistance offered by the walls of such a fine canal to a current of endolymph must be very considerable. Again, a segment of the circle through which the endolymph would pass in circulating round and round was occupied by the utricle, in which during rotation of the body the endolymph would move at a rate different from that of the endolymph in the canal proper. That is to say, the fluid in the utricle would also hinder an even circular circulation of endolymph. He reminded them of the description given of this circulation during and after rotation. It had been said that at the beginning of rotation the endolymph moved more slowly than its containing walls; then, as rotation was continued,

it moved at the same rate as if, in Bárány's phrase, the walls and the endolymph "were frozen together"; and, finally, when rotation was abruptly stopped, the endolymph continued its movement, and so induced the nystagmus which followed rotation. The nystagmus was thus looked upon as entirely dependent upon the movement of endolymph relative to its canal; beginning when the endolymph began to move, stopping when it moved in unison with its canal, beginning again in the opposite direction, when the movement of endolymph was reversed. The physical difficulties he had described, however, made it hard to imagine how such a free round-and-round circulation of endolymph could take place. The objections in the second group had been raised by Bárány as a result of his rotation experiments. Bárány had found that the duration of nystagmus after rotation did not vary in proportion to the duration and speed of the rotation to which an individual was subjected. It was about the same after five complete turns as after twenty. He had found, also, that not only did the duration of the nystagmus vary considerably in different individuals, but that it varied also in the same individual at different times. Finally, he had occasionally observed what he termed an "after-nystagmus" following rotation. This was a nystagmus following upon, and in the opposite direction to, the ordinary nystagmus after rotation. It was difficult to account for these variations on the assumption that the nystagmus was wholly due to endolymph-movement. Bárány, therefore (and with him the speaker agreed), had adopted Abel's theory that the nystagmus was induced by the hyperstimulation of the vestibular nerve-centres in Deiter's nucleus as a result of an abnormally powerful irritation of the end-organ in the ampulla. The variations in duration, intensity, &c., of the nystagmus were therefore attributable not to the continued or interrupted flow of endolymph, but to the exhaustion of one of two opposing nerve-centres. The speaker asked Mr. Scott whether he had employed Bárány's method of measuring the duration of after-nystagmus. It might be asked: What did this powerful stimulation of the fibrillæ which set up nystagmus consist in? The speaker said that he had formed a theory of the function of the narrow part of the canal which would explain this point. He regarded the patent canal beyond the ampulla as providing a means of relieving pressure in the ampulla. During ordinary movements of the head the moderate and momentary rise of ampullary pressure thereby induced was at once lowered by an escape of endolymph from the ampullary to the narrow end of the canal, and, the pressure at the two poles of the utricle being thus equalized, the fibrillæ so rapidly resumed their neutral station that no nystagmus or vertigo resulted. During rotation, however, when the pressure would be considerable and protracted, the frictional resistance offered by the walls of the fine canal would prevent a flow of endolymph through them with sufficient freedom to relieve the pressure in the ampulla, and a hyper-excitation of the nerve-centres, with consequent nystagmus, vertigo, &c., from the prolonged deflection of the fibrillæ would result. So that, in place of looking upon a rapid flow of endolymph through the canal as the cause of nystagmus, he was of opinion that that phenomenon was due to the tardiness of such a

current. He (Dr. McKenzie) noted Mr. Scott's pronouncement that rotation nystagmus was precisely the same "in type and directional characters" when both labyrinths were intact as when only one was functional. But he also reminded the Section of Bárány's invariable finding that when one labyrinth was destroyed the after-nystagmus to the sound side was stronger and lasted longer than that to the affected side. He also referred to Ewald's observation¹ that, in the case of the external canal, movement of endolymph from canal to utricle induced head- and eye-movements more extensive than when the endolymph was moved in the contrary direction, and that the opposite was observed in the case of the superior and posterior canals. Turning to the results of thermal, or caloric, stimulation of the vestibular end-organ the speaker reported that he had failed to obtain an invariable reversal of the nystagmus when the patient was inverted, although both Mr. Scott and Bárány had succeeded in doing so. With healthy individuals in the upright position he had never failed, using the cold test, to obtain the typical nystagmus towards the opposite side. But in other positions of the body the results varied considerably. He had tested many with the head inverted so that the vertex of the skull was facing the ground, and in this position, using the cold test, he had obtained nystagmus towards the opposite side much oftener than towards the same side. He had also tried Bárány's plan of inverting the patient while the nystagmus was in progress, but had again failed to induce a reversal. Occasionally the nystagmus would stop, and this he ascribed partly to the passive congestion of the head and partly to the stimulation of the canals in consequence of the altered position. This variation in his results contrasted so markedly with what was seen in the erect position that he was inclined to question whether the nystagmus was due to a cooling or heating of the endolymph, and consequent production of convection currents. Here, also, some people had experienced a difficulty from the point of view of physics. They doubted whether cold, for example, could affect the outer wall of the labyrinth across the tympanic cavity when that cavity was filled with air, which was a non-conducting medium. Perhaps, however, the explanation was that the change of temperature in the meatus was conveyed to the labyrinth through bone. Reverting to the theories of caloric nystagmus, he suggested that the nystagmus which followed thermal applications might perhaps be due to the direct action of changes of temperature upon the nervous apparatus in the ampullæ. In support of this theory he reminded them of the nystagmus which was induced by galvanism. When, for example, the anode was introduced into one meatus, there was nystagmus to the opposite side, and when the kathode was employed the nystagmus was directed to the same side. These differences were ascribed to the retarding and accelerating influence of the positive and negative poles respectively, and it was possible that cold, like the anode, retarded the transmission of impulses, while heat, like the kathode, encouraged the transmission.

¹ J. R. Ewald, "Physiologische Untersuchungen über das Endorgan des Nervus Octavus," Wiesbaden, 1892, p. 264.

He (Dr. McKenzie) expressed his agreement with Mr. Scott in referring the nystagmoid ocular movements to alternating impulses, the first from the labyrinth causing the slow movement, and the second, from a higher centre, causing the quick movement. The resemblance to the "movement of pursuit" was irresistibly suggestive. Probably the impulse which induced the short movement, though emanating from a centre higher than that governing the slow movement, was purely reflex and independent of the conscious retinal image, since Bárány had reported that the subjective movement of objects did not always correspond to the retinal image of the slow ocular excursion. It usually did so, however. He, like Sir Victor Horsley, admired the ingenuity Mr. Scott had shown in his theory of carotid pulsation. It accounted in the simplest and most beautiful fashion for phenomena which had hitherto been unexplained, and, like the missing part of a complicated puzzle, had placed each fact in its proper place. But at the same time, there were several awkward questions to be answered before the *advocatus diaboli* would be silenced. One of these questions was: if the carotid impact was responsible for the normal stimulus emanating from the canalicular system, would not compression of one carotid always induce nystagmus and vertigo as a result of the withdrawal of the normal stimulus of that side? Again, during moments of violent cardiac excitement, as well as during periods when the cardiac beat was unusually feeble, nystagmus and vertigo should be present. Moreover, supporters of the theory that the nystagmus and vertigo following removal of the labyrinth of one side were due to persistent irritation of the severed nerve-trunk could quote as analogous the experience of a man who had lost a leg by amputation complaining of pain referred to the toes of the amputated member. Vertigo was the pain of the vestibular system. Referring to the allusion, by Mr. Scott, to a case of fistula in the external canal, in which nystagmus to the same side was observed, the speaker drew attention to the rarity of homolateralization of the nystagmus under these circumstances, and asked Mr. Scott if the other ear was quite healthy. There was no inherent reason against a fistula producing nystagmus to the same side, but the speaker had not seen or heard of it before. Before closing, the speaker urged the advisability of the adoption of a uniform terminology in order to facilitate the comprehension of this difficult subject. For this reason, although it was an etymological barbarism, he recommended the use of the term "after-nystagmus" for the nystagmus which followed rotation, because when authors used the word "nystagmus" only, especially when discussing the physiological problem, it often took some little time and trouble to discover whether nystagmus during, or after, rotation was intended. In conclusion, Dr. Dan McKenzie said that he would welcome a discussion by the Section on the diagnostic value of the labyrinth tests, particularly with reference to the necessity for operation. The subject was one of great importance, and he, personally, would be very glad to contribute the results of the little experience he had gathered on the subject.

Mr. A. L. WHITEHEAD asked whether Mr. Scott had observed true rotatory nystagmus, such as occurred in miners. He believed there was only a partial rotation in labyrinthine nystagmus. He had intended to ask a question on the point Sir Victor Horsley mentioned, as to the movement of the head. He had at present under his care a woman who had an acute labyrinthine attack two months ago, with nystagmus, vomiting, vertigo, &c. She was now much better, but he could elicit, by syringing with cold water, a tremendous nystagmus to the right (the lesion was on the left) and, associated with it, clonic movements of the right arm and leg. He asked whether Mr. Scott had noticed any associated movements in the cases on which he had experimented. What Mr. Scott had said concerning compression of the carotids was new to him, and it was only since he got the paper a few days ago that he had had the opportunity of testing the matter. He tested it in one normal case, and also in the case to which he had just alluded. In the normal person the compression seemed to have some effect on the nystagmus, but it did not stop it. In the pathological case the compression did not affect the nystagmus.

Mr. MACLEOD YEARSLEY said he was glad Dr. McKenzie had mentioned the difficulties in the way of accepting movement as taking place in the endolymph. That matter had been drawn attention to by Yves Delage in the *Archives de Zoologie* for 1900, who pointed out that the semicircular canals were practically capillary tubes. That authority emphasized the difficulty in accepting movement of their fluid content by the fact that in the Elasmobranch fishes the endolymph is replaced by a gelatinous substance. Professor Delage also elaborated the theory of co-operant canals, but time would not then permit one to go into his opinions.

Mr. WEST said that he had experience of the care and accuracy of Mr. Scott's methods, and the majority of the observations he had been able personally to confirm; his remarks would therefore be made from the standpoint of a frank acceptance of the clinical data. As to their explanation, there was such a convergence of evidence in favour of the theory of endolymphatic currents that the most likely path of advance was in the acceptance of that theory as a basis. What was the essential cause of such currents? Changes in velocity when a canal was moved in a straight line were not followed by vertigo or nystagmus; a rotatory movement was necessary to produce the difference of pressure between the two ends of the canal, without which there could be no flow created. At a given rate of change of velocity of rotation, the two factors in producing this were the radii of rotation of the two ends of the canal, which were directly proportional to the tangential velocities, and to the positive or negative acceleration of the two ends. The rate of flow would be determined by the difference of the squares of these values, which gave finally a stimulus (proportional to the rate of flow), which varied directly as the radius of rotation of the canal at its centre. Mr. West took exception to the explanation offered of the clinical fact that rotation in one direction was more rapidly efficient as a stimulus than in the other; it was physically demonstrable that the period in which "uniform movement of the endolymph"

was established must be the same in either case. He offered another hypothesis as the explanation, that the degree of deflection was determined by the rate of flow over the hairs of the crista, and that this was actually more rapid after brief rotation when flow took place from canal through ampulla to utricle than in the reverse direction, owing to the formation of a fluid vein through the mass of endolymph in the ampulla in the one case and not in the other; this was illustrated by the comparison of the ampulla and body of the canal with the barrel and nozzle of an ordinary syringe, in which under suction a jet of fluid was projected up the centre of the fluid in the barrel. Finally, he wished to emphasize the fact that there must always be "slip" between the endolymph and the canal-wall, however brief the movement of the head, and that for a given rapidity of movement this was at its absolute maximum at the instant of commencement of the movement; the shortness of the movement had thus no influence on the efficiency of the stimulus. He thought it extremely improbable that the hairs were ever deflected by their own inertia independently of any movement in the fluid, as Mr. Scott seemed to imply, for that would necessitate a considerable difference in the density of the hair from that of the surrounding fluid; and in any case the movement of fluid must of necessity always take place in all rotatory movements of the head, and itself provided a sufficient cause of deflection.

Dr. H. J. DAVIS asked whether the nystagmus was the cause of the vertigo, or the vertigo the cause of the nystagmus. In the discussion the two terms seemed to be used synonymously, but the phenomena were not the same.

Dr. URBAN PRITCHARD said he thought those who were opponents of the physical rotation theory were apt to forget—in saying that the semicircular canals were very small, and that there was a difficulty in accepting the current theory—the exceeding delicacy of the cilia: the least disturbance would be appreciated by them.

Dr. DUNDAS GRANT said it did not seem by any means obvious to him that the impulse conveyed from the semicircular canal towards the ampulla should be more vigorous than that from the utricle towards the ampulla. He would have thought that in view of the greater bulk of fluid and the larger space in which it could move there was a stronger impulse conveyed from the utricle towards the ampulla. The orifice of communication between the utricle and ampulla was also enormously greater than that between the canal and ampulla, and, moreover, the minute calibre of the canal seemed to him to exclude the possibility of any marked flow taking place in it. He thought that the impulse from the utricle to the ampulla would account for some of the phenomena which had been described; for instance, the composite spontaneous nystagmus after unilateral labyrinthectomy was the same as that produced and conveyed from the utricle to the ampulla. The result of pressure on a fistula in the semicircular canal behind the ampulla also supported this opinion. He suggested that the effect of quinine and salicylates upon the labyrinth should

be tested by carrying out some of the experiments mentioned, before and after the administration of these drugs to patients, as helping to decide the question as to the anodyne action of these remedies on the nerve mechanism of equilibration.

The PRESIDENT asked whether the difficulty in obtaining vertical nystagmus was not in part due to the fact that the superior and posterior canals were to some extent antagonistic. He was prepared to accept Crum-Brown's view on the question—namely, that the posterior on one side corresponded to the superior on the other, and vice versa. One must assume that the normal stimulus was from the canal to the ampulla. There was also the question whether, assuming that physical method of stimulation, the superior on one side could be stimulated without the posterior, because they were connected on the non-ampullary side. In studying vertigo it was important to get intelligent patients to say what was the apparent direction of movement of surrounding objects, starting with the simple proposition that when one rotated from right to left the result of cessation was apparent movement of objects from left to right. By investigating apparent movements one could get an idea as to the condition of the horizontal semicircular canal. He failed where the question of vertical movements occurred, because patients had not been able to differentiate movements, *e.g.*, from right below from those from left below, and seemed to describe such movements as vertical. Another question which occurred to him many years ago was, how was it that the semicircular canals were situated in such a position that every auditory stimulus must also stimulate the endolymph and utricle, and therefore the semicircular canals? The caloric nystagmus, as explained by Bárány, was a physiological anomaly. Could one bring oneself to believe, except by a metaphysical process, that there was actually cooling through the tympanic membrane and through the labyrinthine wall? He did not think it possible. A more plausible explanation was that the application of cold caused a cessation of labyrinthine action, and heat caused an increase of it. That view was shared by others also. Another point was how to explain the nystagmus caused by looking out of the window in a moving train. He assumed that the nystagmus associated with vertigo might possibly be an attempt on the part of the eyes to accommodate themselves to the position of surrounding objects. Rotation from right to left caused an appearance of movement of surrounding objects from left to right; it was an attempt to follow moving objects. The result of what he had said was to make him agree with what Sir Victor Horsley stated—that other things besides the nystagmus must be studied, especially the attitude of the patient and the direction of the apparent movement of objects, and that that would enable useful additions to knowledge to be made.

Mr. SCOTT, in reply, thanked Sir Victor Horsley for his remarks, which he greatly valued. He said he had notes of forced movements of the head, trunk, and limbs; such movements only occurred when violent nystagmus was produced by stimulation of the labyrinth. He had concentrated his attention upon

the ocular movements in order to make his observations accurate. To make his views clearer, as they differed somewhat from Mr. Waggett's, he thought it necessary to emphasize the distinction between the physical conditions at the beginning of head-movements and the conditions resulting from the sudden arrest of rotation; in the former case he held that there was no current, and the nerves of the canals in the plane of the movement were equally stimulated by what he had called "inertia deflection"; in the other case—i.e., after rotation—currents would be set up, and he maintained that unequal stimulation was possible. For the sake of distinction he had used the expression "momentum deflection" to indicate the character of the stimulus. He was glad to hear Dr. McKenzie's criticisms, and he hoped he would some day submit a paper to the Section embodying his views. He thought, however, that "nystagmus after rotation" was probably preferable to "after-nystagmus" in English phraseology. He would account for some of Dr. McKenzie's results disagreeing with his own and with Bárány's by presuming incomplete inversion of the head. He (Mr. Scott) was not at variance with Bárány as regards the *degree of intensity* of nystagmus evoked by rotation when one labyrinth was functionless. In the note appended to Table II he had said, "Given the appropriate angular velocity . . . rotation will induce the same *type* of nystagmus with the same *directional* characters" . . . and later on, under Section 9, "Theoretical Data of Rotation," he had more fully considered the matter. With respect to the duration of nystagmus to which Bárány was said to pay so much attention, he (Mr. Scott) had come to the conclusion that provided the type of nystagmus was recognizable its duration was not an infallible guide to the diagnosis of lateralization of the lesion, and he preferred to make this diagnosis only after taking into consideration all the circumstances of the case. With respect to the state of "the other ear" in the cases he had mentioned in his paper, this was examined in detail, and was normal. In addition, he would say there was no evidence in any of cerebellar disease. Replying to Mr. Whitehead, he did not maintain that compression of the common carotid artery provoked nystagmus in every person. His hypothesis explained the causation of nystagmus in certain cases. He had frequently failed to obtain any results from compression: a number of factors were necessary. He expected to reinforce his observations on this point. Of miner's nystagmus, which Mr. Whitehead described, Mr. Scott had had no experience. The type of rotatory nystagmus with which he was familiar was characterized by a much smaller arc of excursion than that which Mr. Whitehead described. He had been sometimes asked how he would explain "coarse nystagmus to one side and fine nystagmus to the other." He was unwilling to express an opinion unless he had seen the case in which this condition was said to exist, but if pressed he would venture to suggest that possibly the "coarse nystagmus" was a horizontal or oblique movement, while the "fine" nystagmus was really the shortest excursion of a rotatory movement, which, when very slight, would appear to be horizontal. Since writing his paper he had seen a man with bilateral otitis media who had lost his left labyrinth, had well-marked

spontaneous horizontal nystagmus to the right and slight rotatory (clockwise) nystagmus to the left. He was at first much exercised by this case, which seemed to upset the hypothesis which he had promulgated, for the nystagmus was not the type which he attributed to carotid pulsation—in fact, compression of the right carotid artery appeared to increase the intensity. Moreover, the right labyrinth reacted to the various tests and the nystagmus was uninfluenced by attempted meatal compression or rarefaction. Useful hearing in the right ear was retained, and there was no loss of bone conduction. The symptoms were explained by the presence of a fistula of the external semicircular canal, covered with granulations. The Eustachian tube being open, perfect meatal compression had not been obtained, air escaping freely into the pharynx. He had passed a tube into the attic to produce antral compression, but that had proved unsatisfactory, the air escaping by the side of the tube. Yet the type of nystagmus clearly pointed to a fistula behind the external ampulla of the remaining labyrinth. (See "Extra-Ampullary Lesions.") He could best answer Dr. Henry Davis by stating that he had seen cases of vertigo during the paroxysm when the patient fell, and no nystagmus could be observed, though sought for. Giddiness provoked by the caloric tests often preceded the nystagmus; in other cases nystagmus could be produced in patients who denied any sensation of giddiness unless the nystagmus was well marked. In a few cases of long-standing spontaneous labyrinthine nystagmus the sensation of giddiness had disappeared and the patients seemed inured to the unusual eye-movements.¹ Dr. Grant's suggestion relating to the investigation of the influence of quinine and salicylates was a valuable one. To those who had some difficulty in conceiving movements of fluid in such narrow canals as the membranous semicircular canals undoubtedly were, he would suggest that the capillaries were far smaller in sectional area, and yet they permitted the flow of more viscid fluid containing solid corpuscles. He was glad Dr. Urban Pritchard had drawn attention to the extreme delicacy of the fibrillæ, for it was really a question of the *relative* size of the fibrillæ to the calibre of the ampullæ and canals. Replying to the President, Mr. Scott thought the paper really dealt with the point concerning Professor Crum-Brown's theory, which, in the light of his observations, he maintained needed modification. According to the present hypothesis, if the fibrillæ of *both* superior ampullæ were deflected equally in the same direction no ocular movements resulted; but if the fibrillæ of *either* or *both* inferior (*i.e.*, posterior) ampullæ were deflected vertical eye-movements took place. He was glad the President would reconsider this point. He had been told by Dr. Duncanson² that when a boxer received a knock-out blow on the side of the point of the jaw he fell down dizzy, and horizontal nystagmus had been observed. When the blow was delivered upwards on the point of the jaw, producing a violent jerk of the head backwards,

¹ Compare valuable paper on "Vertigo," by Rudolph Pause, of Dresden, *Arch. of Otol.*, New York, 1902, xxxi, p. 467.

² "The Knock-out Blow and the Point of the Jaw," by James Gray Duncanson, *Brit. Med. Journ.*, 1903, i, p. 782. Dr. Sherrington had also made observations on this subject.

vertical nystagmus had been noticed. Mr. Scott maintained that these observations corroborated his own. With regard to the President's question about nystagmus noticed in persons looking at, say, telegraph poles, while travelling in a train, McKendrick had pointed out its resemblance to labyrinthine nystagmus. Mr. Scott's opinion was that it was not labyrinthine nystagmus, for it occurred in normal persons when the head was kept still. He regarded it as a pure retinal reflex action of pursuit, which he had described. With respect to the President's difficulty in accepting the theory of convection currents, Mr. Scott had considered the possibility of cold and of heat stimulating the labyrinth, but the observations on inversion of the head dismissed such explanations; for the same reasons the possibility of increased labyrinthine pressure by expansion from heat and vice versa must be rejected. He saw difficulties in the physical explanations of the effects of rotation, but he thought Breuer's theory of convection currents was satisfactory.

Otological Section.

May 8, 1909.

Dr. PETER McBRIDE, President of the Section, in the Chair.

A Specimen of Caries of the Temporal Bone, the Destruction of the Bone closely simulating the Appearance seen after the Performance of the Radical Mastoid Operation.

By A. L. WHITEHEAD, B.S.

DISCUSSION.

Mr. WAGGETT said he had had three instances of the kind in his hospital practice, two of them in one patient. He hoped to bring them on a future occasion.

Dr. DUNDAS GRANT said he had seen the condition in several instances, and in each case it was due to the development of cholesteatoma, which had caused the erosion of bone, almost exactly simulating the appearance of a radical mastoid operation, as in the present case.

Mr. A. CHEATLE said he did not think such cases were very rare.

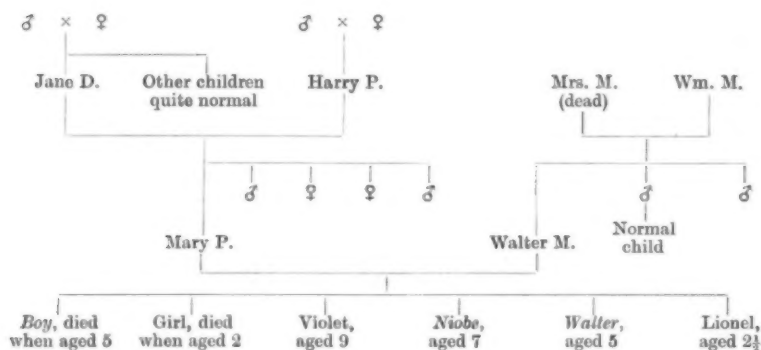
Mr. WHITEHEAD replied that there was chronic otorrhœa on both sides, and the patient died of meningitis, the infection being from the other side. The excavation of the temporal bone was the most complete he had ever seen, the whole of the bridge having been entirely destroyed.

Case of Spontaneous Occurrence of Congenital Deaf-mutism in Three Members of one Family without any previous history of a similar condition.

By W. H. BOWEN, M.S.

Two children, N. M., aged 7, and W. M., aged 5, were brought to the Royal Ear Hospital on March 2, 1909, to be examined with a view to remedying the deafness present. They were found to be absolutely

deaf and quite unresponsive to any sound. No cause for this deafness was found, and no history of disease in the past was ascertainable. The external ears, meatus, and membranæ tympani were normal. The children were sensible, clean, and apparently healthy in all other respects. They had no signs of congenital syphilis. The mother, who brought them from Wilts, stated that her eldest boy, who died from an epidemic fever when aged 5, was also entirely deaf, and was stated to be incurably so when seen at the Royal Ear Hospital at the age of 3. She stated that her husband was a strong, healthy man, that she herself never suffered from illness, that she never had a miscarriage, and that in the family, where the matter had been discussed, the occurrence of the deafness was inexplicable. The father and mother were in no way related.



Note.—The affected members of the family are printed in italics.

DISCUSSION.

Dr. DUNDAS GRANT said the cases seemed to be congenital, and it would be interesting to hear if there was any evidence of failure of development of the vestibular portion of the labyrinth at the same time. In cases of deaf-mutism it was well to make the tests by rotation at all events, and by syringing also if possible, to see whether the labyrinth was active as evidenced by nystagmus.

Dr. DAN MCKENZIE said the subject of the vestibular reactions in deaf-mutism had been investigated by a German writer, Brock, a résumé of whose paper appeared in the *Journal of Laryngology* for 1907.¹ Mr. Tweedie had also dealt with the matter.²

¹ *Journ. of Laryng.*, xxli, 1907, p. 658.

² *Journ. of Laryng.*, xxiii, 1908, p. 592.

Mr. MACLEOD YEARSLEY said the interest of the case was that three such instances occurred in the family without any previous history of any kind. He asked whether Mr. Bowen had inquired about tubercle, insanity, and alcoholism in the histories.

Dr. FITZGERALD POWELL said it would be interesting to learn the family histories further back, especially those of grandfather and grandmother and their collateral members.

Mr. A. CHEATLE said he did not think the absence of the history of deaf-mutism proved it was not a case of hereditary deaf-mutism. Dr. Kerr Love, in his book, showed that several generations were sometimes skipped, and that made the connexion almost impossible to trace. He would like to know if there were any remnants of hearing.

Mr. BOWEN, in reply, said he did not test the patients for vestibular conditions. The mother was a very intelligent woman, and was quite sure there had been no previous illness. A little boy died when aged 5 from scarlet fever. He did not see where such a case could be classified on the Mendelian basis. It was almost unknown for an abnormality to skip more than two generations. He had inquired as far as the great-grandfather of one branch and the grandparents of the other, and there was no history of deaf-mutism. The occurrence of deaf-mutism in families had been dealt with by Professor Karl Pearson and Dr. Jobson Horne, and they showed that whenever a normal person was born in the family he handed on the normal inheritance. The occurrence of spontaneous deaf-mutism in a family was sometimes related to insanity, but in the present case no trace of it could be found, and the aetiology was to him inexplicable.

Notes of a Case of Cholesteatoma of External Auditory Meatus treated by Local Application of Enzymol.

By ADOLPH BRONNER, M.D.

THE patient, a very nervous girl, aged 10, had been deaf, with repeated attacks of earache for about two years. Both meatuses were full of debris. After prolonged syringing a large cholesteatoma was removed from the right meatus. The use of peroxide-of-hydrogen drops greatly facilitated the removal. The child refused to have the left ear syringed. Enzymol drops (1 to 3) were used for fourteen days, and much debris came away. When seen again the left meatus was open and the drum could be seen.

DISCUSSION.

Dr. DUNDAS GRANT asked what enzymol was, and whether the drum when seen was absolutely normal, because the cholesteatomata of the external auditory meatus were often continuations from the external attic or some other cavity of the middle ear. He was disposed to congratulate the child on its not having the ear syringed. Patients often experienced great pain through syringing in cases of cholesteatoma of the external auditory meatus, if the cases were treated as instances of impacted cerumen.

Dr. BRONNER, in reply, said the child was very delicate and had heart disease, and that was why chloroform was not given. The drum could be seen; the case was one of cholesteatoma of the external meatus. The condition was not very common. Enzymol was an American preparation, issued by Fairchild, but he did not know its composition. He had also used it in chronic eczema of the external meatus, where there was necrosis of tissue.

**Protrusion of Synovial Membrane through Tympanic Plate
and beneath Skin of External Auditory Meatus.**

By RICHARD LAKE, F.R.C.S.

THE patient presented himself as an out-patient, complaining of deafness and of a peculiar sound as of moving fluid when eating. On examination there was found on the lower and inner aspect of the anterior meatal wall a small prominence, white and lenticular in shape, having more the appearance of an exostosis, but on investigation it was found to be soft, and its origin being suspected, the patient was made to open and close the jaws whilst the swelling disappeared and reappeared. The cyst is now less tense than formerly. A preparation showing dehiscence in the external meatus was exhibited.

DISCUSSION.

The PRESIDENT (Dr. McBride) said he had never seen anything like it before, and he would be interested to hear whether others had.

Dr. H. J. DAVIS said that when the patient was asked to inflate his own ears by Valsalva's method the sac bulged out like a ballooned membrane. Unless there was some connexion with the middle ear he did not see why this should happen.

Mr. WEST said the only condition he had seen simulating the present one, showing the possible connexion between the synovial membrane in this case and the meatus, was the appearance presented after removal of the anterior

wall of the bony meatus, under which circumstances the lax capsule of the joint might bulge and recede with movements of the jaw.

Mr. WHITEHEAD said he could not satisfy himself that it moved on inflation; he did not detect any difference.

Mr. YEARSLEY said the case added one more to the causes of entotic tinnitus.

Mr. HERBERT TILLEY said he thought the explanation was that a patient frequently closed his jaws tightly when he inflated his cheeks, and if the present patient did so a projection would be caused in the meatus.

Mr. A. CHEATLE said the projection could be controlled by pressure on the joint.

Dr. FITZGERALD POWELL said he thought Mr. Lake had had other similar cases. He showed a specimen in which the posterior wall was partially wanting, and he would like to ask him if he had seen the condyle of the jaw from which the specimen was taken, and whether there was any alteration in the shape of the condyle, as that might possibly account for the opening in the posterior wall.

Mr. LAKE replied that when the man first came under observation he obviously had arthritis, and that was the cause of the cyst being more tense.

Case of Epileptiform Attacks of Labyrinthine Origin.

By RICHARD LAKE, F.R.C.S.

THE patient, a woman, aged 38, had suffered from middle ear suppuration after scarlet fever in infancy. There is no discharge now, but bilateral deafness with tinnitus persists. Vertigo occurs when she stoops and is particularly well marked when either ear is syringed; objects usually appear to move towards the left. Attacks occur without direct stimulation. Both membranes are perforated, the left being almost destroyed. Paracusis Willisii noticed.

The patient gave the following account of the attacks: They commence with a feeling of nausea in the epigastrium which extends up to the head, and vertigo sets in; at about the same time she loses vision as to external objects, but sees flashes of light, and becomes partially unconscious, but knows what is taking place around her. She is much fatigued for the remainder of the day. The caloric test produced identically the same symptoms and, when applied to the left ear, lateral nystagmus when looking to the right. The left pupil is much dilated.

DISCUSSION.

Dr. DUNDAS GRANT said he had a case which very much resembled the present one, in which cholesteatoma was certainly present. The fits entirely ceased upon the removal of the ossicles, this allowing of the escape of the debris.

The PRESIDENT (Dr. McBride) asked whether hysteria had been absolutely excluded in the case, as it was a condition which occurred from time to time in association with ear disease. When a lesion was present in a hysterical patient there was difficulty in deciding whether the attacks were primarily due to the hysteria or to the ear.

Mr. LAKE, in reply, said he only brought forward the case as being one of a large group of cases which were described by Sir William Gowers some time ago as epileptic fits with auditory auræ. He brought it forward to show that it was the ear which was at fault. The condition which followed disturbance of the labyrinth was epileptiform, not epileptic. He would not further prove his contention, except by repeating that by syringing out the ear with cold water he could reproduce the clinical symptoms. He did not say it was not hysteria, but it did not strike him as being such. He believed it was to be explained by the pathological condition of the labyrinth, and seemed to be another illustration of Sir William Gowers's interesting series of cases and from a different point of view.

Intractable Ulcer of Tragus and Fistula of Helix.

By W. H. KELSON, M.D.

THE patient was a girl, aged 6. Her mother says she did not notice anything wrong at birth nor up to a year ago, but then a pimple appeared in front of the left ear, which festered and discharged yellowish matter, and has never completely healed, though scraped and cauterized by a surgeon. On examination an ulcer the size of a threepenny-bit was found involving the left tragus and extending forward from it and covered with a yellowish crust. There was no history or evidence of discharge from the meatus.

DISCUSSION.

Dr. H. J. DAVIS asked whether it was a case of lupus, for it certainly looked like it.

Mr. A. CHEATLE said that, many years ago, he published a similar case of congenital aural fistula in the *Archives of Otology*.¹ An abscess formed and

¹ 1897, xxvi, p. 188.

burst, leaving an ulcer which took a long time to heal up. A fistula remained, which had to be dissected out before a cure was obtained.

Dr. FITZGERALD POWELL said that a similar case in a child, with ulcer and sinus occupying the same position on the face, had been under his care. It proved most intractable and difficult to cure, recurring again and again after curetting, excision, &c.; on each occasion it healed up after the treatment, but again broke out. He had excised the ulcer and dissected the walls of the sinus out, with the same result. Conjunctival reaction with tuberculin was obtained. Treatment by tuberculin injection was tried, when the ulcer healed. He had not seen the case for twelve months, so presumed that it remained well. He thought this case of Dr. Kelson's was probably tuberculous.

Dr. KELSON, in reply, said he brought it forward because he thought there was a catch about it. It looked like lupus or tubercle, but on passing a probe he found it definitely connected with a fistula in the helix, which would not heal. Besides, much honey-like substance could be pressed out of it, such as was found in congenital fistula, which he thought was the cause of it. Possibly it might have become tuberculous, but the simpler explanation seemed to suffice. He had opened up the fistula and dissected out its walls.

Further Notes on Two Cases of Carcinoma of the Ear.

By C. E. WEST, F.R.C.S.

BOTH these cases were included in the short summary recently published in the report of the February meeting of the Otolological Section.¹ Since that date fresh developments have occurred.

Case I.—S. S., male, aged 45, June, 1907, came to the hospital with history of pain in left ear for at least two years; lately slight bleeding. Carcinoma involving all walls of the meatus in the bony part; no glandular enlargement observed. At operation the tympanum was apparently free from disease. Radical mastoid operation; complete excision of the meatus, cartilaginous and bony, removal of tympanic plate made as complete as possible. All apparently well till March 24, 1909, when discharge was complained of. In the anterior part of the cavity there was a fullness, from a minute opening in which serous fluid, mixed with epithelial debris, could be squeezed; no surface ulceration. The opening led into a considerable mass of easily penetrated tissue, a piece of which, on removal and examination, proved to be squamous-celled carcinoma. At a further operation there was found extensive growth passing forward deep to the neck of the lower jaw into the internal

¹ (No. 5), p. 34.

maxillary region, along the line of the Eustachian tube to the neighbourhood of the lateral wall of the pharynx, and upwards through the bone, (?) the root of the greater wing of the sphenoid, and the dura mater, into the brain. Its removal was necessarily abandoned as impossible.

Case II.—W. E., male, aged 68. Operation, October, 1908. Carcinoma involved the right external meatus, the parotid gland, and masseter muscle. Extensive enlargement of cervical glands; irremovable mass in the right side of the root of the tongue. Local removal, to avoid ulceration, of the meatus, parotid, and masseter, together with the proximal lymphatic glands. Squamous-celled carcinoma. This patient was shown at the February meeting. There has been no local recurrence which could be observed; recently there have developed an enormous number of subcutaneous nodules and masses, with some in the muscles; the largest are in the muscles of the left upper arm and left scapula. The glands in the neck have much diminished in size, and the tongue is more freely movable; owing to the limitation of movement of the jaw it is impossible to accurately examine the root of the tongue, but from the absence of pain it is probable that there is no ulceration. When last seen this patient's condition was very bad, very feeble and anæmic; but he was free from pain except in one of the masses which appeared to be affecting the periosteum of the left humerus.

DISCUSSION.

The PRESIDENT (Dr. McBride) said the absence of discussion on the cases was simply because the condition was so rare, but he was only expressing the feeling of the Section when he thanked Mr. West for having brought them forward. Such cases could not too often be brought before their notice.

Dr. BRONNER said he did not doubt that these cases were carcinoma, but some time ago he had a case which showed the typical symptoms of epithelioma of the external meatus, and an expert microscopist declared it to be such. The man was advised to have the growth removed, but he did not turn up. A few days later he got pustular eczema all over his body, and the meatal tumour disappeared absolutely. Some of the cases published as cured epitheliomata were possibly not that disease at all.

Mr. WEST, in reply, said he was almost inclined to apologize to the meeting for bringing these cases forward again; but as he had made statements about them at a previous meeting, and important happenings had occurred with regard to two of them since, he felt bound to bring the additional facts forward. In the ward note on the first case, it was stated that at the time of the original operation there was a pale, isolated, firm granulation at the mouth of the Eustachian orifice. Seeing how the disease had extended, he thought that

was malignant tissue, though at the time it was thought not to be. That raised the point as to the direction of flow of the lymphatics of the mucosa of the tympanum. He would be glad if anyone could tell him whether the lymphatics of the mucosa of the tympanum drained along the Eustachian tube in the submucosa towards the pharynx. If so, it meant an extension of the operative sphere by the removal of the tube in cases of carcinoma of the ear. He was uncertain whether the disease had extended upwards through the root of the greater wing of the sphenoid or the anterior part of the horizontal plate of the squamous. He scraped away as far as he could and grafted the cavity, and the grafts took very well. But since then there was increased fullness in the depth of the cavity, and there was extension going on underneath the cutaneous lining. He was now dying of the symptoms of cerebral compression, and he thought there was a large mass in the temporo-sphenoidal lobe of the brain. There was now loss of memory, difficulty in finding words, and intermittent coma. That man's condition, at the time of the second operation, was exactly similar to the cases described as squamous carcinoma of the tympanum. When he first saw the patient, there was no doubt it was carcinoma of the meatus. It emphasized his contention that the majority of the cases started in the meatus, and he had found that confirmed in the literature of the subject. Such cases seemed to be commoner than he had thought. Though some of them were labelled "Carcinoma of the Tympanum," their description pointed, sometimes explicitly, to their meatal origin. In the second case the only point of interest was the extreme rarity of the dissemination of squamous-celled carcinoma in the subcutaneous tissues. Authorities whom he had consulted seemed to regard this mode of dissemination as practically unique.

Epithelioma of Middle Ear in a Young Man aged 35.

By H. J. DAVIS, M.B.

THE patient was first seen in October, 1908, by Mr. Lake, who diagnosed the condition. As his own beds were full he asked me to take him into the West London Hospital, and this I did.

The history is a very short one. Intense pain in the left ear of three months' duration; no history of a blow; a small granulating polypus was protruding at the meatus. This, in addition to some parotid swelling, was the only indication of the disease. There were no glands.

On November 12 I dissected the auricle forwards, excised the meatus and concha, and removed the whole of the contents of the middle ear, which was eroded down to the internal carotid, which could be seen (and can still be seen) pulsating at the bottom of the wound. The disease proved extensive. The post-aural wound healed rapidly, the deeper parts granulated, and all pain ceased: patient left the hospital with

instructions to report himself once a fortnight. This he failed to do, "as he was quite well."

Three months later he returned with a recurrent growth. The parts were very freely curetted and cauterized with Paquelin cautery, and ten days later he was sent to the X-ray department for treatment. In a month he developed dermatitis. The treatment was discontinued and the disease rapidly returned. The cavity, which is a large one, was again curetted, the masses of growth removed being enormous. The patient is now emaciating, the parotid is involved, and the left facial nerve is paralyzed. He is, however, free of pain, and X-ray treatment is again renewed with a small focusing tube passed into the wound. Discharge of parotid fluid through the ear when at meals seems the only inconvenience. The cavity is lightly plugged with gauze. The youth of the patient and the virulent malignancy of the growth, together with the rarity of the disease, are of interest.

DISCUSSION.

The PRESIDENT (Dr. McBride) said that not the least interesting feature was the enormous extent of the operation cavity.

Mr. WEST said he was much struck by the condition of the cavity, and its similarity to his own first case after the first operation. The growth extended in the same direction, and if one curetted firmly in the present case he thought the growth would be found to extend nearly to the pharynx, and forward into the zygomatic fossa. He expected it went up to the brain also. It was a hopeless case with regard to operation.

Dr. H. J. DAVIS, in reply, said he thought the case bore out Mr. West's idea as to the origin of these cancers, and that the growth probably had started in the meatus and had spread backwards into the middle ear. The X-rays had some influence on the growth. After the rays were discontinued it at once recurred. The internal carotid artery had been exposed eight months, and it could be seen and felt pulsating at the apex of the wound. It was surprising that it had not yet ruptured, as it appeared involved in the growth.

Notes of a Case of Left Temporo-sphenoidal Abscess of Otitic Origin.

By H. J. DAVIS, M.B.

THE patient was a powerful man, aged 24. He was brought to the West London Hospital by two policemen on the evening of April 15. They stated that he "was staggering about the street and spinning

round, and they thought that he had taken poison." There was no evidence of this, and as the patient "seemed to have earache" I was asked to see him. He was sitting in the Casualty department holding his head and crying childishly, and evidently in intense pain. Cerebration slow; he was very giddy with "rushing noises in the head," and he then stated that he had had earache for several days, but no discharge, and had never had earache before. The left meatus was tightly plugged with cerumen and there were no localizing signs whatever. On extracting the cerumen with difficulty, caseous matter was detected; pulse 100; temperature 104° F. Radical mastoid operation forthwith. Middle ear and antrum, a mere slit, were full of pus; mastoid sclerosed. The tegmen was eroded and an extradural abscess evacuated; this was quite local. Bone removed over considerable area; brain not pulsating; dura opened and brain incised; gush of fluid but no pus, brain then pulsated freely. Gauze drain; wound left open, dura stitched. Temperature fell to normal and patient quite rational, though irritable; he was very hungry. Five days after this, temperature rose and patient was sick for the first and only time, very noisy, and "banging his head about the bed," twitching of face, and both legs drawn up; no optic neuritis. Second operation: A large flap turned down and bone removed anteriorly; a temporo-sphenoidal abscess was evacuated. The abscess was at the tip of the lobe and, as far as I could see, in no way connected with the original site of the antral disease.

The patient improved very much and seemed to be doing well, but thirty-six hours later the dressings and pillow were soaked with blood, and arterial blood was found to be streaming from the wound. Under continual irrigation the bleeding points were detected and secured, the tissue round the abscess being soft and necrotic. The hæmorrhage came from large cortical vessels and was profuse. Pulse on returning to the ward 124, and temperature 101° F. Rectal saline (a pint) with adrenalin was given, and the patient recovered, but he was very noisy and it took three men to restrain him; he kept standing up in bed and shouting, and there were certainly no signs of aphasia. Next day patient suddenly became drowsy with symptoms of basal meningitis; left pupil dilated, right pin-point eyes deviated to the left with spontaneous nystagmus to same side; neck rigid. Patient gradually sank and died ten days after admission.

Post-mortem: Left cerebral hemisphere intensely congested; right, normal tissue round abscess cavity almost fluid, the rest firm. The abscess was superficial and extended almost to the tip of the lobe.

Dura mater over antral area firm and adherent to the brain. Posterior fossa; pus oozing from internal auditory meatus; no abscess in cerebellum; but in left posterior fossa dura adherent and suppurative meningitis. This evidently spread through the labyrinth; sinuses free; no thrombi.

The interest in the case lies in the fact that:—

(1) There was no history of old ear affection at any time.

(2) The meatus was tightly plugged with cerumen, which probably cost the patient his life, allowing no exit to the pus at the onset of the disease.

(3) Pus in the antrum retained under pressure gave rise to three different centres of infection: an extradural abscess over the seat of disease; a temporo-sphenoidal abscess apparently unconnected with petrous bone; and, later, a purulent posterior basal meningitis limited to one side. Symptoms only noticed twelve hours before death.

(4) There were no localizing signs whatever till the day the patient died.

A specimen of the temporal and part of the occipital bone of the above case was exhibited.

DISCUSSION.

The PRESIDENT (Dr. McBride) said one of the interesting features seemed to be the forced movements which were observed. He believed they were described as spinning-round movements. Presumably they were labyrinthine, as the lesion was not in the cerebellum.

Mr. WHITEHEAD pointed out that in the notes it was stated that the temporo-sphenoidal abscess was apparently unconnected with the petrous bone. Did Dr. Davis mean that he did not regard the abscess as secondary to the temporal-bone disease, or merely that there was a layer of apparently healthy brain cortex between the abscess and the meninges, as was not uncommonly found in these cases?

Dr. H. J. DAVIS, in reply, said the patient was brought up by the police as a supposed case of poisoning, as he was found staggering about the streets. In reply to Mr. Whitehead, he meant that there was no stalk connecting the temporo-sphenoidal with the extradural abscess, and there was nothing to suppose he had anything beyond the original trouble found at the first operation. With the plug of cerumen tightly wedged in the meatus he must have been in very great pain. It was very difficult to arrest the secondary hæmorrhage, as it came from the deep vessels in the brain. The disease later spread through the labyrinth and internal auditory meatus into the posterior fossa, and the patient died of suppurative basal meningitis, though he had no signs of this when he first presented himself ten days before.

PROCEEDINGS
OF THE
ROYAL SOCIETY OF MEDICINE

VOLUME THE SECOND

COMPRISING THE REPORT OF THE PROCEEDINGS FOR THE
SESSION 1908-9

PATHOLOGICAL SECTION



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1909

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PATHOLOGICAL SECTION.

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The Council think it right to state that the Society does not hold itself in any way responsible for the statements made or the views put forward in the various papers.

Pathological Section.

October 20, 1908.

Mr. S. G. SHATTOCK, President of the Section, in the Chair.

An Investigation into some Aspects of the Action of Arsenic.

By P. N. PANTON.

THE drug used in these experiments was arsenious oxide (As_2O_3) and not the apparently inactive metal; but the custom of referring to the oxide under the name of the metal is so universal that the term "arsenic" has, for the most part, been retained in this paper.

The scope of this investigation includes the changes produced by arsenic in the blood and blood-forming organs of some animals as well as in some of their more important viscera, the part played by arsenic in the production of fatty degeneration being especially considered; in addition numerous experiments were made upon the direct action of arsenic on the circulating blood, particularly with regard to the phenomena of phagocytosis and chemiotaxis.

The investigation is for convenience divided into two parts; in the first part are considered the changes produced in the blood and viscera of rabbits by the administration of arsenic; the second part deals with observations upon phagocytosis and chemiotaxis.

PART I.

In the following experiments arsenic was administered to rabbits by the mouth, subcutaneously, and by intra-peritoneal inoculation. The administration took the form of one or more poisonous doses, or of a considerable number of smaller doses extending over periods of from one week to ten weeks. Eight rabbits were thus treated, four receiving

fatal doses of arsenic and four repeated small doses; in the case of those receiving small doses the arsenic was given once daily; in those animals to whom arsenic was given by the mouth a stomach tube was passed to ensure retention of the full dose. To two additional rabbits phosphorus was given in poisonous doses as a control to the arsenic experiments.

The solution of arsenic employed was a 1 in 120 dilution of arsenious oxide in distilled water to which a trace of dilute hydrochloric acid had been added to ensure complete solution. In order to avoid repetition the results obtained are considered collectively under various headings, and the complete account of the changes induced in any one animal is omitted.

GENERAL OBSERVATIONS ON NUTRITIONAL AND OTHER CHANGES.

Although no special investigations on nutrition were made, a record was kept of the alterations in the weights of the rabbits used, and these were mainly immature and growing animals. Three rabbits were treated with arsenic over periods extending from seven to ten weeks, and in each case the body-weights increased from the time the animals were under observation until the commencement of the administration of the arsenic, when a steady fall in weight took place, followed by a gradual recovery if the dose was not unduly increased. In another experiment two rabbits about 7 weeks old were taken from the same litter, and arsenic was given by the mouth to the heavier animal in considerable doses for eight days, at the end of which time it had lost as much in weight as the other animal had gained, namely, 200 grm. This uniform loss of weight in rabbits under the influence of arsenic is in agreement with Professor Delépine's observations on the feeding of rats on arsenical beer and water in considerable doses [8].

No particular attempt was made to procure immunity to the drug, nor was any obtained; rabbits, indeed, appear to vary greatly in their resistance to arsenic; one animal died a few hours after the administration of 1 gr., another was given 3 gr. of arsenic, and thirty-six hours later seemed perfectly well and died suddenly some hours after a further 3 gr. dose had been given; another rabbit which had been given smaller doses of arsenic for ten weeks died from the effects of $1\frac{1}{3}$ gr. given in $\frac{1}{3}$ gr. doses over four days. None of the rabbits under prolonged treatment can be said to have had optimal therapeutic doses, $\frac{1}{60}$ gr. daily being usually chosen as the initial amount in animals weighing about $1\frac{1}{2}$ kilos, a high proportion to the body-weight.

In the case of one rabbit which was received from a particularly disreputable "fancier," with numerous large and offensive ulcers about the legs and body, rapid healing took place when the animal was placed on arsenic; on the other hand, an animal which was being given arsenic hypodermically developed a local abscess and had to be killed, and it was found that the arsenical solution readily became contaminated by a turbid growth; however, an attempt to grow the colon bacillus and the *Staphylococcus aureus* in a 1 in 300 solution of arsenic failed, but a feeble growth of a diphtheroid bacillus was obtained.

No observations were made upon the nervous system in this investigation and in no case was any clinical indication of peripheral neuritis obtained; the only instance of paralysis of the hind legs occurred in the solitary animal kept as a control.

OBSERVATIONS UPON THE BLOOD AND BONE MARROW.

The blood of four rabbits which received large doses (from 1 gr. to 3 gr.) was examined before and after administration of the drug; these are referred to as the "acute" cases. Three of the rabbits which received repeated doses were given from $\frac{1}{60}$ gr. to $\frac{1}{30}$ gr. at first, and the amounts were gradually increased up to $\frac{1}{12}$ gr., $\frac{1}{8}$ gr., and $\frac{1}{4}$ gr. In these "chronic" cases the blood was examined on two or three occasions before the administration was begun and about once every ten days while under the influence of the drug. In two of the chronic cases considered here the arsenic was given by the mouth, and in one hypodermically.

The Fresh Blood.—The crenation of the red cells and the feebleness of rouleaux formation in varying degrees in the normal rabbit render difficult the recognition of pathological conditions of the kind. In no case, however, did any marked changes take place; in one of the chronic cases rouleaux formation was unusually well shown before the administration of the arsenic, and continued to be present throughout the treatment in equal degree.

The Red Cells and Hæmoglobin.—The number of red cells to the cubic millimetre showed considerable variation in the normal rabbits, whereas the hæmoglobin percentage maintained a very constant level at about 65 per cent. On the whole a fairly steady but not greatly marked diminution took place, both in the number of the red cells and in the percentage of the hæmoglobin; as a rule, this diminution varied directly with loss in body-weight and increase in the dose of arsenic given.

In rabbit 1:—

Average of three examinations before administration of arsenic gave: Number of red cells per cubic millimetre, 5,300,000; hæmoglobin percentage, 65; weight, 1 kilo 490 grm.

After six weeks' treatment: Number of red cells per cubic millimetre, 4,581,250; hæmoglobin percentage, 55; weight, 1 kilo 150 grm. Dose of arsenic, $\frac{1}{16}$ gr., hypodermically, daily.

In rabbit 2:—

Average of three examinations before administration of arsenic gave: Number of red cells per cubic millimetre, 5,500,000; hæmoglobin percentage, 65; weight, 1 kilo 930 grm.

After eight weeks' treatment: Number of red cells per cubic millimetre, 4,962,000 (lowest count obtained); hæmoglobin percentage, 50 +; weight, 1 kilo 910 grm. Dose of arsenic, $\frac{1}{16}$ gr., by mouth, daily.

In rabbit 3:—

Before giving arsenic: Number of red cells per cubic millimetre, 8,000,000; hæmoglobin percentage, 65 +; weight, 1 kilo 900 grm.

After two weeks' treatment: Number of red cells per cubic millimetre, 6,550,000; hæmoglobin percentage, 65 +; weight, 1 kilo 800 grm. Dose of arsenic, $\frac{1}{16}$ gr., by mouth, daily.

After five weeks' treatment: Number of red cells per cubic millimetre, 5,012,500; hæmoglobin percentage, 65 +; weight, 1 kilo 910 grm. Dose of arsenic, $\frac{1}{16}$ gr. daily.

After eight weeks' treatment: Number of red cells per cubic millimetre, 4,900,000; hæmoglobin percentage, 65; weight, 1 kilo 380 grm.

(Only one examination was made before giving arsenic and three during the administration.)

(A hæmoglobin percentage number, such as 50 +, means over 50 per cent. and under 55 per cent.)

In the interval between the last two examinations the rabbit became ill and weak and the arsenic was for the time omitted. The rabbit received altogether about 7 gr. of arsenic.

In the stained blood in the three cases no increase in the number of red cells showing polychromatophilic degeneration was observed. An occasional normoblast was seen both before and after the administration of the arsenic; on only one occasion were as many as eight normoblasts seen, while counting 500 white cells. It appears, therefore, that the only changes produced in the number of red cells and percentage of hæmoglobin was a decrease below the normal, which was accompanied by evidence of ill-health in the rabbit; in other words, the changes were those of an ordinary and mild secondary anæmia such as might be produced by a variety of causes.

The Leucocytes.—The number of leucocytes to the cubic millimetre varied in the different animals, but these variations were inconsiderable

in extent and in no way attributable to the action of the arsenic. Numerous differential counts, in each case of 500 cells, were made, and the relative numbers of the white cells remained very constant. No increase of the eosinophile or other granular cells was obtained, nor did any of the bone marrow cells appear in the peripheral blood. There were only two exceptions to the above statements. In the case of one rabbit, which died twenty-two hours after the administration of 1 gr. of arsenic by the mouth, the normal blood showed 32.2 per cent. of polynuclear neutrophiles and 62.4 per cent. of small lymphocytes, the post-mortem blood (taken from the heart) showed 74 per cent. of polynuclear neutrophiles and 16.5 per cent. of small lymphocytes; in the other acute cases no such increase of the polynuclear neutrophiles at the expense of the small lymphocytes was observed; the bone marrow cells of this rabbit consisted almost entirely of mononuclear non-granular cells. In the case of the rabbit which was being given arsenic hypodermically, the white cells rose suddenly to 30,000 per centimetre, and of these 87 per cent. were polynuclear neutrophiles; a considerable local abscess was discovered and the animal killed; over 75 per cent. of the white cells of the bone marrow were found to be granular cells.

The Bone Marrow.—A remarkable condition of the bone marrow has been ascribed by Stockman and Charteris to the action of arsenic; they found an extreme hyaline degeneration of this tissue with great diminution in the number of marrow cells. One of the plates illustrating their account shows a section of the marrow in which the cells have almost entirely disappeared and the tissue has been replaced by a hyaline substance. They do not consider, however, this change to be peculiar to arsenic poisoning, and attribute their failure to reproduce it in rabbits and dogs to an absence of emaciation in their animals [7]. No such changes were observed in any of these cases. The shaft of the femur invariably contained red marrow of normal naked-eye appearance, nor was there any alteration in the size of the medullary cavity of the bone. Those rabbits which had received arsenic for a considerable period were markedly wasted. In each case a portion of the marrow was taken from the shaft of the femur, slide smears were made, and the films stained by Leishman's stain. In one of the chronic cases sections were made and stained with carbol-thionin and with hæmalum and eosin. Both red and white cells appeared as numerous in the smears as in health. In the sections made a few strands of oedematous fatty tissue were seen lying between the cells (oedema and other evidence of cardiac failure were seen in sections of the other organs), but no suggestion

was obtained of any hyaline or other form of degeneration of the marrow. It is difficult to estimate the results of the differential counts done upon the bone marrow films. The relative percentages of the cells in the normal marrow of the rabbit are given as:—

Lymphoid cells	51.3
Granular cells	48.4
Nucleated red cells	11.7

(PRICE JONES)

Considerable variations from the average, however, occur, and only marked and constant alterations could be considered as evidence of any particular pathological change. The various granular and non-granular cells were differentiated in these counts, but no constant variation was seen in any one variety of cell, and there is no object in reproducing the detailed enumerations. The following are the general results:—

In rabbit 1 (chronic):—

Granular cells	75.8 per cent.
Non-granular cells	24.2 "
Nucleated red cells	22.0 "

The high preponderance of granular cells in this case is accounted for by the subcutaneous abscess which had developed.

In rabbit 2 (chronic):—

Granular cells	60.0 per cent.
Non-granular cells	40.0 "
Nucleated red cells	31.2 "

In rabbit 3 (acute):—

Granular cells	39.2 per cent.
Non-granular cells	60.8 "
Nucleated red cells	17.6 "

In rabbit 4 (acute):—

Granular cells	12.0 per cent.
Non-granular cells	88.0 "
Nucleated red cells	5.6 "

In rabbit 5 (acute):—

Granular cells	18.0 per cent.
Non-granular cells	82.0 "
Nucleated red cells	5.8 "

In rabbit 6 (acute):—

Granular cells	5.0 per cent.
Non-granular cells	95.0 "
Nucleated red cells	0.8 "

In the last case exceptionally few red cells were present in the films. On the whole, there seemed to be an increase of the granular cells in the chronic, of the non-granular cells in the acute cases. The nucleated red cells were increased in the chronic and decreased in the acute.

Considerable care was taken in estimating the blood-changes in this investigation, particularly in view of the contradictory statements made upon the action of arsenic on the blood and bone marrow, and also in the hope of throwing some light upon the remarkable, though temporary, effect produced by arsenic in cases of pernicious anæmia. In this latter respect such evidence as has been obtained is mainly of a negative character; the absence of any constant or characteristic change produced in the blood or bone marrow would seem to indicate that the drug does not act by stimulating new blood-formation. The effects produced by arsenic in pernicious anæmia would rather seem to resemble the results of arsenical treatment in such parasitic diseases as trypanosomiasis, and the absence of any specific action of the drug on blood-formation is some evidence in favour of the parasitic theory of the causation of pernicious anæmia.

OBSERVATIONS UPON SOME GENERAL CHANGES IN THE VISCERA.

Free Iron Reaction.—A microscopical examination of the viscera for the presence of free iron was made in each case. In one of the chronic and two of the acute cases no reaction was obtained in any of the viscera. In the remaining four cases a free iron reaction was obtained in the spleen, and in two instances, namely, one acute and one chronic case, the reactions were particularly well marked. In only one case was a free iron reaction obtained in the liver, and that was a feeble one; in the remaining viscera the reaction was invariably negative.

Vascular Changes.—Death usually occurred suddenly, and in the majority of post-mortems the heart was found in diastole, with both ventricles greatly dilated. This, in common with other vascular changes, was considerably more marked in the acute cases. Evidence of death by cardiac failure was an almost constant feature in the visceral sections of all cases, and in some instances an excess of fluid was found in the serous sacs and more particularly in the peritoneal cavity. The vessels of the liver invariably showed great vascular dilatation, the liver cells were in a condition of cloudy swelling, and in two examples there were areas of necrosis. The vascular changes in the kidney were equally

marked, the vessels were distended with blood, and bleeding had taken place both into the tubules, in some instances, and into the glomeruli. The vascular spaces in the spleen were greatly distended in the acute cases, and in one of the chronic cases there was fibrosis of the organ. The other viscera examined showed vascular dilatation in equal degree, and occasional hæmorrhages were found in their substance.

Fatty Changes.—The powerful action of arsenic in producing fatty degeneration is so widely recognized that it is only necessary to give one frequently repeated quotation in support of this effect of the drug: "A widespread fatty granular degeneration, again, is characteristic of arsenical poisoning. It affects the liver and kidneys, the intestinal epithelium, and voluntary muscular fibre. The degeneration is sometimes as marked as that met with in phosphorus poisoning" (Oliver) [6]. This feature of arsenical action, indeed, has given rise to the suggestion that the widespread fatty degeneration met with in pernicious anæmia is produced, not by the disease, but by the drug used in its treatment. By giving to rabbits doses of arsenic greater in proportion to their body-weight than are probably ever taken by man it was expected that a well-marked fatty change would result. In these experiments no reliance was placed on the naked-eye appearance of the viscera, but portions of every important organ were fixed in formalin, cut in gum, and stained with Sharlach R, and practically no evidence of fatty degeneration was observed. In two of the chronic and one of the acute cases blood-films were taken during life, fixed in formalin vapour and stained for fat, in each case with a negative result. Those animals which had received prolonged arsenical treatment were wasted and showed diminution in the subcutaneous, mesenteric, and perinephric fat. In no case could the fat present in the liver be said to be above normal limits; in some instances there appeared to be an actual diminution in the amount of the liver fat. A chemical analysis of two of these livers was kindly made for me by Dr. Mavrogordato, who informed me that "the fat came out to 13.7 per cent. of dry substance, which is within normal limits." Sections of the stomach and small and large intestines were only examined for fat in two instances; no fatty change in the epithelium was found.

The spleen and thymus gland were examined in most instances and with a negative result. The kidneys were examined in each case and with one exception no fat was found. In one of the acute cases a few fat droplets were found in two or three portions of the tubules, but the amount of fat present could be passed as within the normal limits. No fat reaction was obtained in the muscular portion of the diaphragm in

any case. The cardiac muscle fibres were, with the exception of one case, entirely free from fat droplets. One rabbit had been given arsenic in considerable doses for about ten weeks and had obviously suffered in nutrition from the effects of the drug; it was finally given a fatal dose of $1\frac{1}{3}$ gr. spread over three days, a method which was thought most suited for the production of advanced morbid changes. In this rabbit fatty change was found in the cardiac muscle; large areas of the sections were quite free from fat, but in several fibres numerous small fatty droplets were present, and this must be allowed to be pathological; no fatty change, however, was present in any other viscus. This animal was in a septic condition at the commencement of the arsenic treatment, but at the time of death the various lesions were healed. The adrenal gland was the only organ in which fat was present to any degree. In the Sharlach sections the cortical portions of the adrenal appeared to consist entirely of fat; a few fat droplets were sometimes present also in the medullary portions. So extensive, however, is the Sharlach staining area in such sections of the suprarenals as I have examined in normal rabbits that I do not consider any abnormal fatty change to have been produced in the rabbits treated with arsenic.

As a control to the above experiments phosphorus was administered to two additional rabbits. One rabbit received $\frac{1}{12}$ gr., $\frac{1}{6}$ gr., and $\frac{1}{3}$ gr. of phosphorus in oil on three successive days, and was chloroformed on the fourth day. The other was given $\frac{1}{3}$ gr. of phosphorus in oil, and died forty-eight hours later. In neither case was any fatty change produced in the diaphragm or cardiac muscle. In both instances, however, the liver showed extensive fatty degeneration. Fatty change was also present in the renal epithelium in the case of one rabbit to a very marked degree. The fat present in the suprarenals did not differ to any appreciable extent from that found in the normal or the arsenicated rabbits.

In a further experiment arsenic was given by the mouth to a rabbit in considerable doses, which amounted to 1 gr. in eight days. At the end of this time it died suddenly and was found to have diminished in weight from 970 grm. to 760 grm. Sections of the heart muscle, diaphragm, liver, and kidney were stained for fat and compared with sections of these viscera from a rabbit of the same age and about the same weight which had been killed under an anæsthetic. In neither rabbit was any fat present in the heart muscle, diaphragm, or kidney, while the liver of the normal rabbit contained considerably more fatty droplets than that of the animal which had received arsenic.

Uncomplicated cases of poisoning by arsenic in man are not of common occurrence and usually fall into the hands of the toxicologist, who is concerned only with the detection of the poison. In the account of the Manchester epidemic described by Kelynack and Kirkby [3] the following statement is made: "Most of the organs and muscles of the body present more or less extensive fatty degeneration," but no details are given of the data upon which this statement is based, and though fatal cases are mentioned in the book, and one fatal case is described, there is no account of any post-mortem examination. In a paper referring to the same epidemic J. C. Muir [5] gives in an appendix short post-mortem accounts of nine fatal cases, but makes little mention of any fatty change, though the liver is described as "fatty" in some instances, apparently from its naked-eye appearance. All these patients, too, had pulmonary tuberculosis, many of them in a very advanced degree, and the quantity of beer consumed by the patients seen during the epidemic "frequently exceeded a gallon a day" (Kelynack). Some fatty change in the liver might thus have been accounted for even if no arsenic had been taken, nor can a naked-eye diagnosis of fatty degeneration in a viscus be accepted as of any value.

It would be unwise to infer from a failure to produce fatty degeneration in rabbits by the administration of arsenic that this drug never produces such a change in the human subject, but there seems to me sufficient indication that this property of arsenic has been greatly exaggerated.

OBSERVATIONS UPON SOME SPECIAL CHANGES IN THE VISCERA.

The Thymus.—The animals used in these experiments were, so far as could be ascertained, young and growing rabbits. Fairly constant changes were found in the thymus glands which were examined. The vascular changes have already been referred to, and these were more marked in those animals which had received large doses, the thymus sections showing large numbers of greatly dilated capillaries filled with blood, and occasional hæmorrhages into the substance of the gland. The following further changes were found in the acute cases: One rabbit died within two hours of receiving the arsenic; the thymus was found to be of normal size, the interlobular connective tissue was not relatively increased, eosinophile cells were numerous, the concentric corpuscles were few and for the most part ill-formed. Another rabbit died twelve hours after receiving the arsenic—this was a fully grown animal of uncertain

age; the thymus was small and atrophic, consisting largely of connective tissue, eosinophile cells were scanty and the concentric corpuscles few and inconspicuous. Another rabbit died within twenty-two hours; the thymus was of normal size, the proportions of lymphoid and connective tissue were about normal, very few eosinophile cells were seen and the concentric corpuscles were scanty and of small size. The last rabbit died thirty hours after the arsenic; the thymus in this animal was small, but there was no increase in the relative proportion of connective tissue, the eosinophile cells were few, and the concentric corpuscles were very few and very small. Of the chronic cases, the thymus of the first animal was unfortunately not examined. The second rabbit was a young animal and its thymus was found to be of about the normal size, but sections showed a considerable increase in the amount of interlobular connective tissue; eosinophile cells were almost absent. Careful examination of several sections failed to reveal a single well-formed concentric corpuscle, though a few badly formed corpuscles were found after prolonged searching. The third rabbit was a somewhat small and immature animal; at the post-mortem no trace of the thymus gland could be found—some gelatinous-looking material was found in the usual site, but sections made of this revealed œdematous fibroid tissue, and no vestige of lymphoid tissue could be found. In the normal animal kept as a control the thymus gland was found to be normal, the eosinophile cells were plentiful, and the concentric corpuscles numerous and well formed. In the two rabbits poisoned with phosphorus the eosinophile cells were somewhat diminished, but the concentric corpuscles were normal in size and number. The thymus of a wasted and ill-nourished rabbit, which had received no arsenic, was examined and was found to be normal, the concentric corpuscles being particularly large and numerous.

In a control experiment two rabbits of about the same size, which were known to be 7 weeks old and of the same litter, were kept under the same conditions. A grain of arsenic was given to one rabbit over a period of eight days, when it died suddenly; the other rabbit was killed. The rabbit which had received arsenic had lost considerably in weight, and the fat throughout the body was diminished; the thymus was soft and extremely small, weighing only 0.3 gm.; numerous small hæmorrhages were present on the surface; on section the lymphoid tissue was found to have been very largely replaced by fibrous tissue; no eosinophile cells were seen and no concentric corpuscles recognized. The thymus of the control animals was normal to the naked eye and on section; it weighed 1.85 gm.

It would seem from the above experiments that arsenic has a very powerful action upon the thymus gland. Even in the cases of those rabbits which died within a few hours of the administration of arsenic certain changes were found in the thymus glands which were absent in those rabbits poisoned by phosphorus, while in the rabbits which received repeated doses the thymus was found to be completely atrophied after some weeks' treatment, and after eight days' treatment wasted to one-sixth of its normal weight and mainly composed of fibrous tissue.

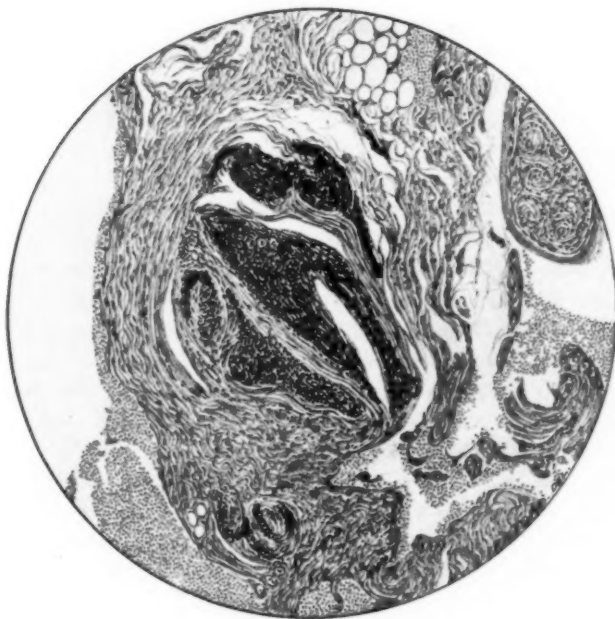


FIG. 1.

A complete transverse section through the thymus of a young rabbit after eight days' administration of arsenic. The section consists mainly of fibrous tissue, fat, and extravasated blood. The darker area in the centre of the field shows the lymphoid cells which remained, lying in cellular fibrous tissue. Drawn under $\frac{1}{3}$ objective; No. 2 ocular.

It must be allowed, however, that the majority of these animals were considerably wasted, and the atrophy of the thymus may be in part accounted for as an accompaniment of the general loss in nutrition.

The atrophy of the thymus in marasmic children has been described by Dudgeon [1], and it is evident from his paper that this may be of very considerable degree; he found, however, in his sections that the concentric corpuscles were apparently more numerous, certainly more conspicuous, than in health, nor does the replacement of lymphoid tissue by other structures appear in his diagrams to have proceeded to the same extent as in the glands investigated in this paper. Apart from the retrogressive changes which take place in the thymus as part of the general result of malnutrition, I conclude that arsenic itself is capable of inducing an atrophy of this organ.

In the *suprarenal gland* no special changes were observed other than the suggestion of excessive fat production, as mentioned before. Little or no pigment or chrome substance was observed in any of the sections.



FIG. 2.

A transverse section of the thymus of a normal control rabbit of the same age as the preceding, showing normal lymphoid tissue. In addition to two blood-vessels full of red cells, there will be seen in the right half of the field two small Hassall's corpuscles. Objective $\frac{3}{4}$, No. 2 ocular.

The other viscera examined were the stomach, intestines, liver, spleen, kidneys, diaphragm, and heart muscle. No special or constant changes in these were observed other than those which have been already detailed.

To sum up, the changes produced by arsenic in rabbits are:—

To cause a diminution in the body-weight if given in sufficient dose.

To produce an ordinary and mild secondary anæmia, unaccompanied by any specific changes in the blood or bone marrow.

To produce little or no fatty degeneration in any viscus, even if given in excessive amounts.

To bring about a condition of cardiac failure and marked capillary dilatation throughout the body.

To induce retrogressive changes in the thymus when given in considerable doses, as evidenced by the diminution of the eosinophile cells and the concentric corpuscles in the "acute" cases and the complete atrophy of the gland in the "chronic" cases.

PART II.

The following experiments deal with the direct action of arsenious acid upon the white cells of the blood, particularly with a view to the alterations produced in the phagocytic activity of the cells by the presence of the drug in strong and in weak solutions. In addition, a few observations are also made upon its influence on the allied phenomenon of chemiotaxis.

TECHNIQUE.

The solutions of arsenious acid used were made, for the most part, by dissolving the substance in normal saline solution made up with tap-water, fresh solutions being used for each experiment. The organism upon which the phagocytic activity of the leucocytes was tested was the *Staphylococcus aureus*, but in a few instances other organisms were employed. As a rule, the arsenious acid solution was added directly to the blood, that is to say, the result of mixing equal volumes of blood, a suspension of *aureus* in citrate solution and a solution of arsenious acid was compared with a mixture of blood, *aureus*, and normal saline solution. Some experiments were also made upon the action of arsenious acid on the washed white corpuscles, as in Wright's method. The effect of arsenic was thus investigated both in strong or poisonous and in weak or therapeutic solutions; the experiments were further extended to a similar investigation of the actions of a few other chemical substances. The injection of arsenical solutions into the peritoneal cavity of animals was done in the usual manner and with aseptic precautions.

In the first series of experiments the alteration in the phagocytic activity of the cells brought about by the addition of arsenic in *strong* solution was investigated. The strength of the arsenic solution given in each case is the actual strength in which the leucocytes were working, that is to say, in most instances one-third the strength of the original solution.

The following "hæmo-phagocytic" indices were obtained in the usual manner; they are chosen as examples from repeated observations:—

- (1) 1 volume of blood, 1 volume of *aureus* in citrate, 1 volume of arsenic solution :
50 cells contain 5 cocci.
1 volume of blood, 1 volume of *aureus* in citrate, 1 volume citrate solution :
50 cells contain 82 cocci.
Strength of arsenic solution, 1 in 300.
- (2) 1 volume of blood, 1 volume of *aureus* in citrate, 1 volume of arsenic solution :
50 cells contain 8 cocci.
1 volume of blood, 1 volume of *aureus* in citrate, 1 volume of citrate solution :
50 cells contain 69 cocci.
Strength of arsenic solution, 1 in 1,500.
- (3) 1 volume of blood, 1 volume of *aureus* in citrate, 1 volume of arsenic solution :
50 cells contain 18 cocci.
1 volume of blood, 1 volume of *aureus* in citrate, 1 volume of citrate solution :
50 cells contain 90 cocci.
Strength of arsenic solution, 1 in 2,700.
- (4) 1 volume of blood, 1 volume of *aureus* in citrate, 1 volume of arsenic solution :
50 cells contain 25 cocci.
1 volume of blood, 1 volume of *aureus* in citrate, 1 volume of tap-water :
50 cells contain 51 cocci.
Strength of arsenic solution, 1 in 6,000.

The following experiments were made to determine whether the action of the arsenic was upon the cells, the serum, or the bacteria: Blood was taken into two tubes, the one containing a 1 in 300 solution of arsenic made up with citrate and saline, the other containing citrate and saline only. The mixture was shaken up and incubated for half an hour. Both tubes were centrifuged, the solutions were drawn off, and the cells washed three times with citrate solution. The phagocytic indices were then obtained in the usual way with the following results:—

- 1 volume of arsenicated cells, 1 volume of *aureus*, 1 volume of citrate, 1 volume of serum :
50 cells contain 9 cocci.
- The cells were unaltered in shape or staining reactions.
- 1 volume of citrated cells, 1 volume of *aureus*, 1 volume of citrate, 1 volume of serum :
50 cells contain 156 cocci.
- 1 volume of citrated cells, 1 volume of *aureus*, 1 volume of arsenic (1 in 300), 1 volume of
serum :
50 cells contain 3 cocci.

The action of the arsenic appears to be upon the cells and to persist after removal of the poison.

That the action of arsenic on the cell can take place in the complete absence of serum is shown by the following experiment:—

Blood was taken into two tubes containing citrated saline solution:

the cells were centrifuged and washed free of plasma. One cellular deposit was then shaken up with an arsenical solution in saline of a strength of 1 in 500; the other deposit was shaken up with normal saline; both were then incubated for fifteen minutes at 37° C. The tubes were next centrifuged, the cells again washed three times in normal saline solution, and the indices of each sample taken. Result:—

Arsenicated cells, <i>aureus</i> , serum :	50 cells contain 18 cocci.
Normal cells, <i>aureus</i> , serum :	50 cells contain 220 cocci.

Exactly similar depressant results were obtained with arsenical solutions of the strength of 1 in 500, 1 in 2,000, and 1 in 7,500. With a dilution of 1 in 20,000, the arsenicated cells were equally active as the normal cells.

An attempt was made to restore the activity to those cells which had been paralysed by the stronger solutions by adding citrate solution to them and replacing them in the incubator. The phagocytic indices were taken at intervals of a few hours, but little or no return to the normal was obtained, and those cells which had originally been treated with arsenic tended to degenerate with greater rapidity than the normal cells.

It is evident from the above experiments that the action of arsenic upon the blood in dilutions down to about 1 in 10,000 is either to prevent or markedly inhibit phagocytosis; in lesser dilution this action is lost. The action is a permanent one upon the cells. It is hardly conceivable, however, that arsenious acid can circulate in the blood during life in a dilution at any rate stronger than 1 in 10,000 even after highly poisonous doses have been absorbed.

The next series of experiments deals with the addition of some other drugs to the blood in *strong* solution and their effect upon phagocytosis. The effect of the hydrochloride of quinine and the perchloride of mercury is shown in the following table:—

1 volume of blood, 1 volume of <i>aureus</i> , 1 volume of citrate :	50 cells contain 69 cocci.
1 volume of blood, 1 volume of <i>aureus</i> , 1 volume of quinine : Strength of quinine, 1 in 300.	50 cells contain 4 cocci.
1 volume of blood, 1 volume of <i>aureus</i> , 1 volume of quinine : Strength of quinine, 1 in 1,500.	50 cells contain 2 cocci.
1 volume of blood, 1 volume of <i>aureus</i> , 1 volume of mercury : Strength of mercury, 1 in 300.	50 cells contain 0 cocci.
1 volume of blood, 1 volume of <i>aureus</i> , 1 volume of mercury : Strength of mercury, 1 in 1,500.	50 cells contain 2 cocci.

The action of these substances is therefore, like that of arsenic, to prevent phagocytosis. It may be noted that quinine is commonly given as an instance of a drug which exerts a negative chemiotactic influence and checks amoeboid movement—paralysing effects which prevent phagocytosis.

A similar series of tests was performed with nucleic acid, the solution employed being that supplied by Parke, Davis. A comparable, but less obvious, inhibition was found to occur, but to be well marked only in strong solution, thus :—

1 volume of blood, 1 volume of *aureus*, 1 volume of nucleic acid :
50 cells contain 26 cocci.
Strength of nucleic acid, 1 in 60.

1 volume of blood, 1 volume of *aureus*, 1 volume of citrate solution :
50 cells contain 69 cocci.

In lesser dilutions little or no effect was observed.

Since sodium citrate is necessarily involved in the technique of all phagocytic reactions, the following experiments were done to determine its share in the inhibition of phagocytosis :—

	1 volume of blood, 1 volume of tap-water, 1 volume of <i>aureus</i> in citrate :	50 cells contain 104 cocci.
	2 volumes of blood and 1 volume of 0.85 per cent. citrate solution were then incubated for fifty minutes, then :	
	1 volume of the mixture, 1 volume of citrate solution, 1 volume of <i>aureus</i> in citrate :	50 cells contain 10 cocci.
Also	Arsenic in 0.85 per cent. citrate, blood, <i>aureus</i> :	50 cells contain 120 cocci.
	Dilution of arsenic, 1 in 150,000.	
	Citrate 0.85 per cent., blood, <i>aureus</i> :	50 cells contain 54 cocci.
Then	1 volume of arsenic in citrate, 2 volumes of blood. Each incubated one hour at 37° C.	
And	1 volume of citrate, 2 volumes of blood.	
And	2 loops of arsenic in citrated blood mixture, 1 loop of <i>aureus</i> :	50 cells contain 36 cocci.
	2 loops of citrated blood mixture, 1 loop of <i>aureus</i> :	50 cells contain 16 cocci.

In another experiment blood was taken into citrate solution, centrifuged and washed thoroughly in normal saline, and—

2 volumes of cells, 1 volume of <i>aureus</i> in saline, 1 volume of serum, 1 volume of tap-water :	50 cells contain 47 cocci.
2 volumes of cells, 1 volume of <i>aureus</i> in saline, 1 volume of serum, 1 volume of citrate 0.85 per cent. :	50 cells contain 19 cocci.

The presence of sodium citrate therefore appears to inhibit phagocytosis to a considerable extent.

Since, in the foregoing experiments the strengths of the dilutions of the various drugs employed were such as could not occur in the circulating blood, except under highly exceptional circumstances, the following attempts were made to determine if arsenic in very *weak* solution, such as might be supposed to exist in the blood of a patient the subject of arsenical treatment, had any influence upon phagocytic activity. The alterations obtained, as was to be expected, were not so marked as those resulting from the stronger solutions; in order to avoid, therefore, the essential errors of technique and "the personal factor," numerous similar experiments were performed and the counts were made and entered upon "unknown bloods." The results, on the whole, were unexpectedly constant, and the following are given as typical examples:—

Equal volumes of blood, <i>aureus</i> , and saline :	50 cells contain 25 cocci.
Equal volumes of blood, <i>aureus</i> , and arsenic in saline : Dilution of arsenic, 1 in 45,000.	50 cells contain 37 cocci.
Equal volumes of blood, <i>aureus</i> , and arsenic in saline : Dilution of arsenic, 1 in 90,000.	50 cells contain 56 cocci.
Equal volumes of blood, <i>aureus</i> , and arsenic in saline : Dilution of arsenic, 1 in 225,000.	50 cells contain 81 cocci.
Also	
1st tube—blood, <i>aureus</i> , saline :	50 cells contain 38 cocci.
2nd tube—blood, <i>aureus</i> , saline :	50 cells contain 54 cocci.
1st tube—blood, <i>aureus</i> , arsenic in saline :	50 cells contain 111 cocci.
2nd tube—blood, <i>aureus</i> , arsenic in saline :	50 cells contain 90 cocci.
Dilution of arsenic in both tubes, 1 in 300,000.	

A similar result was obtained on substituting the *Bacillus coli* for the *Staphylococcus aureus*.

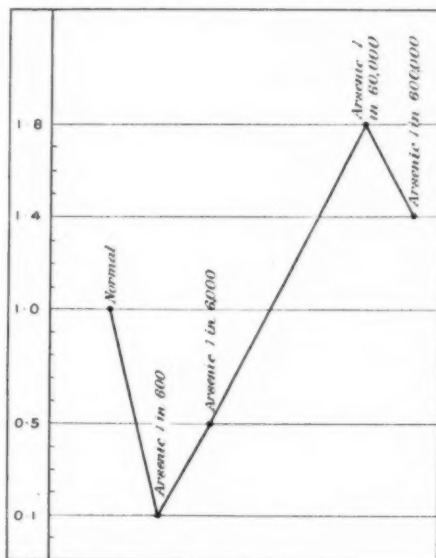
It was found that the most marked stimulating effects were produced by arsenic in dilutions of 1 in 200,000 to 1 in 300,000; in dilutions of less than 1 in 500,000 the effect became diminished. The phagocytic indices, in dilutions of about 1 in 200,000, averaged 2.0 or a little over. The effect on phagocytosis of arsenic in strong and in weak solutions resembles the early and late effects of injecting a bacterial toxin, or the paralysing effect of a strong toxin and the stimulating effect of a weaker one.

If the following series of experiments were placed in the form of a phagocytic curve a negative phase would appear, followed by recovery,

a rise to well above the normal, and again a drop towards the normal, these variations being brought about solely by increasing the dilution of the arsenic :—

Blood, <i>aureus</i> , tap-water (normal) :	50 cells contain 51 cocci.
Blood, <i>aureus</i> , arsenic :	50 cells contain 7 cocci.
Dilution of arsenic, 1 in 600.	
Blood, <i>aureus</i> , arsenic :	50 cells contain 25 cocci.
Dilution of arsenic, 1 in 6,000.	
Blood, <i>aureus</i> , arsenic :	50 cells contain 93 cocci.
Dilution of arsenic, 1 in 60,000.	
Blood, <i>aureus</i> , arsenic :	50 cells contain 71 cocci.
Dilution of arsenic, 1 in 600,000.	

Or described in a curve :—



The following experiments were made in order to determine if the stimulating effect of arsenic was upon the leucocytes themselves. Blood was taken into two tubes, one containing citrated saline solution, the other 1 in 100,000 arsenious acid solution in citrated saline. The two tubes were incubated for one hour at 37° C., then centrifuged, and the

cells were subsequently washed three times in normal salt solution.
Result:—

Normal cells, serum, <i>aureus</i> :	contain 152 cocci.
Arsenicated cells, serum, <i>aureus</i> :	contain 249 cocci.

Three counts of 50 cells each were made, and in each count the normal cells were the less active. The aggregate of the three counts gave :—

Normal cells, serum, <i>aureus</i> :	contain 572 cocci.
Arsenicated cells, serum, <i>aureus</i> :	contain 737 cocci.
Normal washed cells, saline, serum, <i>aureus</i> in saline :	contain 277 cocci.
Normal washed cells, arsenic (1 in 50,000), serum, <i>aureus</i> :	contain 377 cocci.

It therefore appears that the action of arsenic in dilute solution is upon the cells and is not quite so marked as in the experiments in which the arsenic was directly added to the blood. It was found impossible to demonstrate the increased phagocytic activity of the arsenicated cells if serum was omitted from the mixture of cells and cocci, or if the serum was heated before being added. The action of the arsenic on the cells is seen to be sufficiently permanent to outlast the repeated washing and centrifuging of the leucocytes, but the following experiment shows that it disappears after a few hours: The cells were prepared as in the previous experiment, with the exception that the strength of arsenic used was 1 in 200,000, and the mixtures were incubated for only twenty minutes.

Normal cells, <i>aureus</i> , serum :	100 cells contain 156 cocci.
Arsenicated cells, <i>aureus</i> , serum :	100 cells contain 250 cocci.

Then cells were again shaken up in saline and reincubated for one hour at 37° C.

Indices taken about two and a half hours from the commencement of the experiment :—

Normal cells, <i>aureus</i> , serum :	150 cells contain 269 cocci.
Arsenicated cells, <i>aureus</i> , serum :	150 cells contain 265 cocci.

After a further incubation of three hours :—

Normal cells, <i>aureus</i> , serum :	50 cells contain 138 cocci.
Arsenicated cells, <i>aureus</i> , serum :	50 cells contain 124 cocci.

It thus appears that if arsenious acid is added to the blood in dilutions of 1 in 200,000 to 1 in 300,000 an increased phagocytic activity is produced. This increased phagocytosis is due to the action of the arsenic on the cells, and tends to disappear in a few hours if the arsenic is withdrawn. In the presence of the arsenic the cell becomes about twice as active as the normal cell. That the stimulating effect of arsenic on the cells outside the body should be produced by such a dilute solution as 1 in 300,000 is scarcely surprising. Arsenious acid is known to be absorbed into the blood of patients taking the drug either by the mouth or subcutaneously, but I am not acquainted with any analyses showing the strength of the arsenic in the circulation. If, however, we assume that a man of 10 st. has 12 lb., or 84,000 gr., of blood, that he takes $\text{m}\nu$ of Fowler's solution four times a day, and that he can keep in solution in his blood the whole of a two days' supply, the dilution of the arsenic in his blood would be about $\frac{2}{3}$ gr. in 84,000 gr., or 1 in 200,000; so that the artificial dilutions employed may be on a par with those obtained by the administration of the drug by the mouth.

A few phagocytic indices only were estimated on patients, all of whom had been taking arsenic in therapeutic doses for a considerable time. These gave to the *Staphylococcus aureus* :—

A wasting child with secondary anæmia	...	Index 1.2
A case of pernicious anæmia 1.3
A similar case 1.3
A similar case 2.4

A further series of experiments was performed to see if some other chemical substances, which, like arsenic, inhibit phagocytosis in strong dilutions, stimulate it in weak dilutions.

In the case of quinine it was found that if weak solutions of this drug were substituted for normal saline in diluting blood, little if any effect on the phagocytic powers of the leucocytes was produced. The dilutions of quinine employed were 1 in 30,000, 1 in 100,000, and 1 in 300,000.

In the case of nucleic acid numerous observations were made in dilutions ranging from 1 in 600 to 1 in 300,000, and no appreciable difference in phagocytic activity was observed.

The following experiment shows the difference in effects of adding nucleic acid, arsenious acid, and normal saline to the blood. All

dilutions were made with saline solutions and the films were counted without reference to the "Key"—

Blood, saline, <i>aureus</i> :	50 cells contain 48 cocci.
Blood, nucleic acid, <i>aureus</i> :	50 cells contain 48 cocci.
Dilution of nucleic acid, 1 in 30,000.	
Blood, nucleic acid, <i>aureus</i> :	50 cells contain 38 cocci.
Dilution of nucleic acid, 1 in 150,000.	
Blood, nucleic acid, <i>aureus</i> :	50 cells contain 32 cocci.
Dilution of nucleic acid, 1 in 300,000.	
Blood, arsenious acid, <i>aureus</i> :	50 cells contain 72 cocci.
Dilution of arsenic, 1 in 300,000.	

It appears, then, that nucleic acid, in spite of the marked power it exhibits of stimulating the generative function of the leucocytes, is unable, at any rate outside the body, to appreciably increase their appetite. A similarly negative result with nucleic acid has been obtained by Ledingham and Bulloch [4].

In the following experiments arsenic was administered to animals in order to observe the chemiotactic effects produced, as evidenced by the nature of the cellular exudate in the peritoneal cavity which resulted. In one rabbit 3 gr. of arsenic in 10 c.c. of water were injected intraperitoneally, and twenty-four hours later a similar dose was given. The animal died suddenly four hours after the second injection. The peritoneal exudate was blood-stained and the cells in it consisted almost entirely (91.5 per cent.) of endothelial cells. In a second rabbit to whom 2 gr. of arsenic in water were given by the mouth, death took place about twelve hours later, and a considerable amount of blood-tinged peritoneal fluid was found in the abdomen. The white cells in this fluid were very scarce and consisted entirely of endothelial cells. A third rabbit was given 2 gr. of arsenic in strong solution into the peritoneum; death took place within two hours. A slight excess of free fluid was present in the abdomen, and the cells in this consisted almost entirely of endothelial cells.

Two guinea-pigs were injected intraperitoneally into the peritoneal cavity with 1 gr. of arsenic in strong solution, and were dead within two hours. Very little peritoneal fluid was found, and the scanty cells were almost entirely endothelial.

In order to contrast the chemiotactic effects of injecting arsenic in strong and in weak solution, two guinea-pigs were injected intraperitoneally—the one with 5 c.c. of a 1 in 500 solution of arsenic, the other

with 5 c.c. of 1 in 200,000 solution; both solutions were made with normal saline. Both pigs were killed two hours after the injection. In the case of the pig which had received the 1 in 500 solution an excess of deeply blood-stained peritoneal fluid was found in the abdominal cavity. A differential count of the white cells in this fluid gave:—

Polynuclear neutrophiles	2.5 per cent.
Small lymphocytes	32.5 "
Large lymphocytes	1.0 "
Eosinophiles	4.0 "
Endothelial cells	60.0 "
				<hr/>
				100.0

In films taken from the omentum a few endothelial cells only were seen. In the case of the pig which had received the 1 in 200,000 solution of arsenic a considerable excess of faintly turbid but not blood-stained fluid was found in the peritoneal cavity. A differential count of the cells in this fluid gave:—

Polynuclear neutrophiles	87.0 per cent.
Small lymphocytes	5.5 "
Large lymphocytes	1.0 "
Large hyaline cells	3.5 "
Eosinophiles	1.0 "
Endothelial cells	2.0 "
				<hr/>
				100.0

In films taken from the omentum numerous large clumps of polynuclear neutrophiles were found, and these cells comprised 93 per cent. of the total leucocytes present. It is seen that the effect of injecting arsenic into the peritoneal cavity in strong and in dilute solution is strikingly different; in the one case a "passive" exudation results, and in the other case an "active" one. This difference is strictly comparable with the opposing effects on phagocytosis of arsenic added to the blood in concentrated and in weak solutions.

Both in the last experiment and in the other cases in which arsenic was injected in strong solution the cells of the peritoneal fluid closely resembled those normal to the peritoneal cavity, and this may be taken as evidence of the negative chemiotactic influence of the drug in this dilution in so far as an active migration of the blood-leucocytes into the peritoneal cavity was prevented; there is no evidence that any cells were actually repelled from the arsenical area.

In the case of the pig which was injected with the dilute solution, an active emigration of cells took place into the abdominal cavity, but it is difficult to be certain to what extent this was produced by the arsenic alone. It has been shown by Dudgeon and Ross [2] that the injection of normal saline into the peritoneal cavity causes an active exudation of cells, and it is extremely difficult to eliminate this factor; in their experiments, however, the peritoneal cells, two hours after injection of normal saline, yielded 37.4 per cent. of small lymphocytes and 3.41 per cent. of eosinophiles as against 87 per cent. of polynuclear neutrophiles present in the above experiment after the same period, so that on the whole it seems reasonable to suppose that the arsenic, of itself, exercised an attractive influence upon the blood-phagocytes, and that they moved towards the sphere of the stimulating fluid.

To sum up, arsenic, when added to the blood *in vitro*, exercises a paralysing effect upon the leucocytes when given in strong solution, as evidenced by the alteration in the phagocytic activity of the blood; a similar effect is produced by the hydrochloride of quinine, the perchloride of mercury, and to a much less extent by nucleic acid. This action of arsenic is a direct action upon the leucocytes and can take place in the complete absence of serum. When arsenic is added to the blood *in vitro* in very dilute solution the reverse effect is obtained and an increased phagocytic activity is produced. Comparable alterations in chemiotactic phenomena are obtained when arsenic is injected into the peritoneal cavity in strong and in weak solutions.

These experiments with arsenic seem to afford the means of demonstrating in part the actions of an active chemical substance when given in poisonous and in therapeutic doses. It is improbable that arsenic is the only substance capable of producing these effects; a parallel has already been suggested between the results of the varying dilutions of arsenic upon phagocytosis and the effect of a bacterial toxin—at any rate in one aspect of the general action of the toxin; and investigation might show similar reactions produced by other drugs in common use, such as strychnine or alcohol, reactions obvious in their poisonous, vague in their “tonic” effects. It appears to me, apart from any general vasomotor or special local action, that if a recognized “tonic” substance be shown to render a protoplasmic cell twice as active in performing some of its functions as it was before the administration of the drug, one might expect the general result of a general increase in protoplasmic activity to be such as is commonly described as a “tonic” effect.

That such a substance as arsenic is thus found to exercise a recognizable influence, when acting in such extreme dilution as 1 in 200,000, is a practical demonstration of the benefits which may result from the addition of the small quantity of a dilute drug to the considerable bulk of an individual. The increased activity produced in the phagocytes suggests also an explanation of the value of arsenic in cases of suppuration.

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DISCUSSION.

The PRESIDENT (Mr. Shattock) asked the author how far he had examined the bones in such cases for marks of chronic inflammation. At the College of Surgeons there was a curious series of bones from cows which were accidentally poisoned with arsenic. They showed marked periostitis. Obviously, members would be agreed that the most important part of the communication was that concerning the increased phagocytosis. This was so important and suggestive that it almost made one ask whether the whole of the beneficial effect of arsenic in the treatment of disease might not be due to the stimulation of the leucocytes and the consequently increased phagocytosis. For in all cases of disease in which arsenic appeared beneficial, it might be assumed that the pathogenesis was of an infective kind, *e.g.*, trypanosomiasis, pernicious anæmia, lymphadenoma, and leukæmia. He asked whether Dr. Panton proposed to test in a similar way the action of antimony, which was the most recent remedy for the treatment of trypanosomiasis.

Dr. BERNSTEIN asked whether the author had tested the phagocytosis of the blood of the pigs mentioned as having been fed on arsenic. Also, had Dr. Panton done any experiments on patients who had had large doses of

arsenic, such as those suffering from severe anæmia? Such an investigation would be much more valuable than test tube experiments, approaching more to the natural conditions. Would the arsenic in test tube be in the same condition as in the stomach? It would be important to know exactly in what form the arsenic enters the circulation from the digestive tract.

Dr. PANTON, in reply, said the bones of the cases were not examined; only the bone marrow. He did try perchloride of mercury, but not the dilute solution. He had not found what percentage of arsenic was in the blood in the usual arsenic treatment. In a patient taking arsenic in therapeutic doses the dilution of arsenic in his blood might be 1 in 200,000. He had not tried new arsenical preparations in the same way. He did not try Dr. Bernstein's suggestion on the pigs, but he did on a few patients who were having arsenic. But patients with severe anæmia had so few phagocytes. The indices of the half-dozen patients whom he examined varied from 1'3 to 2'4.

A Case of a Rare Disease of the Long Bones terminating in Sarcoma of the Femur.

By R. LAWFORD KNAGGS and O. C. GRÜNER.

THOMAS C., aged 53, was admitted into the Leeds General Infirmary on January 18, 1907. He had enjoyed good health till a fortnight before, when he began to suffer from aching pains in the legs. In the left leg the pain was slight and was felt just below the knee in front. In the right, it was situated in the thigh, behind and to the outer side, and was most acute in the lower part. It was aggravated by walking. He had never noticed any swelling and his doctor had diagnosed "sciatica." On the night of January 17, as his wife was helping him to remove his trousers, the right foot slipped out of the garment and struck the floor. He heard a crack and found his leg was useless. When admitted there was a fracture at the junction of the upper and middle thirds of the right femur. Crepitus was not distinct, but a kind of creak could be occasionally elicited. There was some thickening about the bone for 2 in. or 3 in. above the fracture, but it had never been noticed by the patient. A skiagram showed a fracture with the ends of the two fragments in satisfactory apposition, but no appearance suggestive of new growth. The right lower limb was awkward and clumsy owing to a marked genu valgum and flat-foot. The tibia in its lower third presented a definite swelling not unlike a syphilitic node,

and there was also a slight outward bending here which intensified the effect of the genu valgum. A skiagram of the swelling showed some expansion of the bone and some irregular spaces in its interior which suggested absorption of cancellous tissue.

The patient stated that as a boy his legs were straight, that the genu valgum developed gradually after early boyhood, and the swelling in the lower part of the tibia made its appearance in adolescence. The deformity was supposed to be due to "working" and "shortness of food." The left leg was straight, but there was a little doubtful thickening in the lower third of the tibia and some obvious thickening of the upper third of the femur below the great trochanter. The long bones of both upper extremities, and the clavicles, presented no abnormality. The lungs, the heart, the abdominal viscera, and the urine were healthy. The patient gave a history of great privation up to the age of 18. There was no history or sign of old gonorrhœa or syphilis. He had always been temperate and had only been off work a fortnight in thirty years, and then for "cold."

There was considerable difficulty in treating the fracture owing to the genu valgum, and on March 11, though there was thought to be some attempt at union there was still a good deal of mobility, and a skiagram showed some displacement of the fragments had occurred. On April 2, another skiagram showed much greater displacement and erosion of the ends of the fragments as if by growth. No doubt as to the true nature of the disease was now entertained, and on April 10 the limb was amputated at the hip-joint by the anterior racket incision, the femoral glands, which were subsequently found to be diseased, being removed during the dissection.

The patient made a satisfactory recovery, and prophylactic treatment by Coley's fluid was commenced on May 3. Six minims of Parke, Davis and Co.'s solution produced a definite reaction and was injected about twice a week till the beginning of November, when recurrence had taken place in the iliac glands. A large mass could be felt beneath the abdominal parietes above Poupart's ligament. The patient was unaware of it and his general condition was very good. He had grown quite stout. Death did not take place till some months later.

The femur, tibia, and fibula of the amputated limb were reserved for examination. They were sawn through longitudinally, but not until they had been in preservative fluid for some weeks; consequently they were bleached.

Macroscopic Appearances.—The appearance presented by all three bones was unusual. The cancellous structure was largely replaced by soft tissue which cut easily and was gritty. It spread through almost the whole length of the bones in an irregular manner, and was in the main sharply differentiated from the cancellous bone, which was not replaced by it. Lying in this soft tissue were innumerable calcareous fragments or spicules which, though not preventing portions for examination from being readily cut out with a knife, were yet sufficient to require decalcification before satisfactory microscopic sections could be made. In some parts the calcareous infiltration was so slight that this was not necessary. There were also larger masses of very dense bone which were situated in some instances where marrow is normally present, such as the medullary cavity of the tibia. The tibia and fibula participated in a slight outward bend, and in addition to the swelling at the lower part of the tibia there were two fusiform enlargements of the fibula, but with these exceptions the bones presented a normal contour. The shell of compact bone was quite normal except where it was eroded at the seat of fracture, and there was no distinct subperiosteal thickening.

The Femur.—In this bone the soft gritty tissue reached well into the great trochanter and into the neck to within an inch of the head. It was very sharply margined at this end of the bone and filled in the outer compact shell as low as the fracture. In the lower fragment it was continued into the middle of the femur, but below that a more natural appearance was presented. The medullary cavity filled with ordinary marrow existed in the lower half of the bone, and the lower cancellous end showed no definite morbid change. The cancellous tissue of the head and neck was very dense and sclerosed, and this condition extended quite up to the edge of the disease. Lying entirely within the tract of soft gritty material, $2\frac{1}{2}$ in. below the great trochanter, was a distinctly defined mass of hard bony material 1 in. in diameter. It was firmer and more resistant than normal cancellous bone, but had not the same ring or crisp sound and feel when prodded with forceps. The diseased tissue where it filled in the upper half of the medullary cavity was firm and fibrous-looking and largely impregnated with lime salts. At the seat of fracture the ends of the two fragments were eroded for a short distance on their compact surfaces. There was no expansion of the bone, but the destruction of the compact tissue was complete on the inner side. In the muscle surrounding this portion of the bone there was an infiltrating mass of growth. There was no defined subperiosteal tumour.

The *tibia* showed the same replacement of cancellous tissue and central medulla by soft gritty material from end to end of the bone. It was sharply margined below, but in the upper end the separation was less defined. The diseased tract showed up clearly and its continuity in the upper two-thirds of the bone was produced by a confluence of patches. In the lower 4 in. of the bone the tissue had the elastic softness of cartilage but not its appearance—a condition that explained the evidence of absorption shown by the skiagram. Here also the bone was increased in thickness, but it was difficult to say whether it was due to expansion or to periosteal deposit.



FIG. 1.

From the medullary canal of the tibia. A groundwork of mucous connective tissue in which only a few fat cells are to be seen. (Low power.)

The *fibula* presented similar changes. The lower half of the bone and the upper third were distinctly enlarged, and the two enlargements were connected by a short piece of bone of more natural size and shape.

The compact tissue was thickened in the upper enlargement, but in the lower it looked as if it had been encroached upon by the altered tissue in the interior. The cancellous part of the bone in the lower enlargement was largely filled in with the diseased tissue and measured 1 in. across.

Microscopic Appearances.—In many places where the unhealthy tissue joined true bone the line of separation was sharply defined. To show the immediate transition from health to disease a section was prepared from a block cut from the lower end of the right tibia close

to the articular cartilage. In the area of true bone, which even to the naked eye looked atrophic, slender laminated trabeculae were widely separated by fat marrow in which blood-vessels were present. The latter were more abundant at the edge of the disease. The trabeculae were

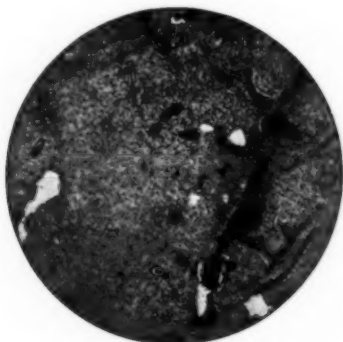


FIG. 2.

From the lower end of the fibula. Active changes in progress; new bone formation and absorption; irregular distribution of trabeculae, with accumulation of elongated cells around; osteoid tissue forms part of some of the trabeculae. (Low power.)



FIG. 3.

A portion of the preceding, under a higher power, showing young bone formation and the character of the surrounding tissue.

quiescent, ossification and absorption being apparently at a standstill, and the tissues generally were singularly free from cells. Some of these quiescent trabeculae could be traced into the diseased part, where active

ossification and absorption were going on. Here the trabeculae were thicker, more irregular, and not laminated, and the spaces between them were filled with well-marked connective tissue. The numerous nuclei stained well and no trace of fat marrow remained. The transition from the fat marrow of the true bone to this intertrabecular connective tissue was abrupt, trabeculae being bounded on one side by fat marrow and on the other by connective tissue, and only slight signs of the invasion of the fat by wisps of connective tissue between gaps in the trabeculae could be seen. The connective tissue was much more vascular than the marrow. This replacement of fat marrow by a well-marked and in many places highly cellular connective tissue, in which irregular ossification was going on, hand in hand with bone absorption, was characteristic of the change in those portions of all three bones which were obviously unnatural.

Sections made from tissue which filled up the medullary cavities of both tibia and fibula were almost entirely composed of this connective tissue groundwork. In both bones at this part it was very largely necrotic, only a framework of myxomatous tissue being present and the nuclei failing to stain. The blood-supply was not deficient and the vessels were plentiful in many places; but bone was not entirely absent, and a circumscribed calcareous area in the section from the tibia showed a close arrangement of trabeculae presenting Howship's lacunae, but an entire absence of cellular elements. In other places a finely granular deposit marked the early traces of an attempt at ossification, which had no doubt been interrupted by cellular death. This tendency to necrosis was also a feature of sections taken from the upper part of the fibula and the upper half of the femur.

At both ends of the tibia and at the lower end of the fibula a very different state of affairs was found. Many irregular trabeculae were scattered about, separated by connective tissue, which was very cellular, especially in the vicinity of bone in which active change was progressing. The trabeculae were of various sizes, and ossification and absorption were going on with great activity. Many osteoclasts were present, and Howship's lacunae could be seen on almost every piece of bone. Osteoclasts and Howship's lacunae were often found close to a row of osteoblasts in process of being transformed into bone cells on the same trabecula. The earliest formation of bone was shown in some sections as a group of calcareous granules surrounding and obscuring a cell in the midst of a field of connective tissue. Not far off might be seen a much thicker mass just acquiring the characteristics of a definite

trabecula, and showing three or four bone cells embedded in a mass of granular deposit. In the trabeculae undergoing active changes the central portions stained with hæmatoxylin and were obviously calcified, whilst the peripheral portions stained with fuchsin (van Gieson) and were not calcified (osteoid tissue). When, in the larger trabeculae, ossification was progressing and a layer of osteoblasts was being included, processes passed out from the bone between the cells and could be seen to blend and be continuous with the connective tissue fibres. These processes stained the same deep colour as the osteoid tissue from which they sprang. It was clear that it was the connective tissue that became calcified.

All the new bone, however, was probably not formed afresh in this way, for the continuity of the abnormal bone with the trabeculae in the undiseased area already alluded to suggested that the latter were not always entirely removed before fresh additions to their mass began to be made.

A section taken from the upper part of the femur showed very similar changes to those already described in the tibia and fibula. The trabeculae, however, were larger, showed some tendency to fuse and to assume a disposition suggestive of an attempt to imitate true bone formation. The connective tissue was much more fibrous and dense, and the cells were large, with deeply staining nuclei, and often lay in spaces formed by the connective tissue. About 1 in. above the fracture another section was in places necrotic, but the connective tissue could be traced until it merged gradually into a well-marked, spindle-celled sarcoma. A nodule of growth in the muscle in the vicinity of the fracture was found to be composed of a similar spindle-celled growth supported in a connective tissue basis identical with that seen throughout the three bones.

The *glands* contained numerous pale-staining cells, with oval nuclei lying in the lymph paths. It was difficult to say whether they were sarcoma cells or proliferated endothelial cells.

In a review of the case as a whole the following points stand out:—

- (I) The definite history of prolonged starvation during childhood and youth.
- (II) The general alteration in the character and structure of the bones; and
- (III) The supervention of sarcoma.

(I) THE HISTORY OF PRIVATION.

The history of privation is of importance because the histological features of the abnormal tissue bear a strong resemblance to those of rickets, a disease generally acknowledged to be due to causes which influence nutrition.

The genu valgum and the swelling in the tibia made their appearance in early boyhood, and were attributed by his friends and neighbours to "want of support." He stated that he was never properly fed till he was aged about 18, when he was able to earn enough to keep himself. Until then his usual diet was "dry bread and treacle, and sometimes not even that." Meat he got perhaps once a week, when some of the neighbours would take pity on him and give him a meal. He illustrated the straits to which the family were reduced, owing to the drunkenness of the father, by the fact that they used to beg for tea-leaves after they had been used, in order to make a second brew for themselves.

There can hardly be any doubt that the alterations in structure which were common to all three bones developed during this period of his life, and that they were in all probability the consequence of deficient nutrition.

(II) THE PECULIAR STRUCTURE OF THE DISEASED BONE.

One of the most striking features is the change in the marrow. A groundwork of connective tissue filling in the spaces between bone trabeculae is met with in several abnormal conditions of bone. It is found in rickets, extending from the periosteum to the central medulla, where the peripheral layers are chiefly affected, the central portion remaining as red marrow.¹ In osteogenesis imperfecta, the congenital condition which is marked by multiple fractures *in utero* or in the first few years of life, the appearance of the marrow is practically the same. It is described as "almost entirely cedematous myxomatous connective tissue" near the epiphysal line "and in the cortical marrow spaces." "Further away from the epiphysis the centre of the marrow spaces is filled with normal marrow separated from the trabeculae by a zone of connective tissue."²

Very similar is the condition seen in osteitis deformans, but in the

¹ Cornil and Ranvier, "Manual of Pathological Histology," 2nd edit., English translation, 1882, i, p. 372.

² "Keen's Surgery," ii, p. 53. Lovett and Nicholls, *Brit. Med. Journ.*, 1906, ii, p. 915.

specimen prepared by us for the purpose of contrast there is no sign whatever of normal marrow, and the connective tissue looks firmer and presents none of the myxomatous appearance seen in this case and described in *osteogenesis imperfecta*.

In a rare disease of bone, named by Messrs. Pitts and Shattock "non-calcifying plastic osteitis," the spaces between the trabeculae "were uniformly occupied by a cellular connective tissue" in which there was an abundant vascular supply.¹ In the case which forms the subject of this communication, and which seems to us to bear a very close resemblance to Messrs. Pitts' and Shattock's case, the connective tissue had taken the place of the marrow, both in the central medullary canals and between the trabeculae which were substituted for the cancellous tissue. It was markedly vascular, and, excepting one or two sections in which a few scattered clusters of fat cells appeared, the replacement of the normal by the abnormal marrow was complete.

Another very noticeable characteristic was the way in which bone formation and absorption were going on or had taken place. The total amount of bone was very much diminished, and its distribution was uneven and irregular. The trabeculae were widely separated by the connective tissue, in which they seemed to be scattered without any obvious arrangement; but evidence was not wanting that as ossification progressed there was a tendency for some degree of orderliness to be assumed. They showed, however, no sign of Haversian systems, and the bone cells were angular and rounded and not stellate, and there were no canaliculi.

In some of the trabeculae when calcification was progressing the central portion was obviously calcareous (staining with hæmatoxylin), and the peripheral portions were devoid of lime salts.

This was very much the same condition as Messrs. Pitts and Shattock described in their case of "non-calcifying plastic osteitis." In rickets, when bone is developing from connective tissue marrow and periosteum, osteoid tissue (by which term is to be understood bone tissue which is not calcified) is first formed, and ossification begins in the central portions of the trabeculae. In *osteogenesis imperfecta* there is no development of osteoid tissue, but otherwise the irregular formation of bony trabeculae is very much the same as in our case. In *osteitis deformans* the formation of bone, instead of being diminished, is excessive. The trabeculae show the same kind of cells and the absence of canaliculi, and

¹ *Trans. Path. Soc., Lond.*, 1897, *xlvi*, p. 180.

are calcareous throughout, but they are much larger and separated by comparatively narrow tracts of connective tissue. There is, however, a very definite but irregular Haversian formation, often found filling in the scalloped side of a trabecula. This apparently originates by processes forming part of a trabecula coming in contact with one another and enclosing lacunæ of connective tissue marrow. Ossification then advances from the circumference until the enclosed marrow has become replaced by bone, sometimes to such an extent that no central cavity remains. Obviously the Haversian arrangement in these morbid conditions of bone is dependent upon the extent to which ossification progresses. And possibly the absence of it in the case under review may have been connected with the tendency for considerable tracts of tissue to pass into a state of necrosis.

The condition of the bone in the immediate proximity of the diseased tissue varied. In the section made to show the transition from health to disease the healthy area was occupied by atrophic laminated trabeculæ. In other parts, noticeably the great trochanter and the neck of the femur, there was marked sclerosis.

(III) THE SUPERVENTION OF SARCOMA.

This is of much interest in view of the resemblance that the case has to osteitis deformans in histological details. It is, perhaps, one of the best known facts about the latter disease that some cases terminate in cancer. The direct relation of the malignant tumour to the disease is not, however, always very clear. Thus in one of Sir James Paget's three cases of malignant termination "a growth corresponding to the growth described as epithelioma of the arachnoid surface of the dura mater" grew from the inner surface of the dura mater. Here the cranium was affected by the osteitis (Case 4). In another (Case 2) a medullary cancer infiltrated the upper part of the right humerus and led to a spontaneous fracture. The rest of the bone was healthy, though the thighs, legs, and spine showed the changes of osteitis deformans. And in the other (Case 1) the upper third of the left radius, which was otherwise healthy, was involved in a mass of cancer with growths of bone extending into it.¹

On the other hand, Messrs. Pitts and Shattock quote a case of Mr. Mackellar's, which they regard as one of osteitis deformans, in which the growth of a cystic fibrifying sarcoma ensued in the upper end

¹ *Med. Chir. Trans.*, Lond., 1877, lx, p. 37.

of the diseased tibia. The age of this man was 58; the ages of Sir James Paget's cases 60, between 50 and 60, and 68.

Osteitis deformans is a disease of later life, and its tendency is to progress and involve bone after bone. In rickets, which is a disease of early life, and is believed to have some connexion with certain benign growths (enchondromata), the tendency is towards cure, and for the abnormal bone and the intertrabecular tissue to be replaced by bone of a natural type (Hektoen).

When these facts are remembered the development of sarcoma in these allied forms of chronic bone disease affecting people in middle or old age is not to be wondered at. The presence of an abnormal tissue in a state of ill-regulated activity, which continues in this condition for many years and shows no tendency to cure, is a very obvious favouring condition.

DISCUSSION.

The PRESIDENT said he supposed that in this case no one would doubt the conclusion that the advent of a sarcomatous change was an accident, that it was not a part of the disease, any more than it was in the case of osteitis deformans. Therefore the question for consideration was what the primary disease was to be classed as. Should it be called a case of osteitis deformans or classed among the group of conditions to which he had given the name of "non-calcifying plastic osteitis"? It appeared to him that the present case was remarkably like the one referred to so frequently by Mr. Knaggs, and exhibited by Mr. Pitts some years ago in conjunction with himself. In that, there was definite localized replacement of the tibia, in its upper two-thirds, by soft spongy new bone. It was exactly the kind of tissue which had been so accurately and minutely described by Mr. Knaggs. In that case it was suggested by the late Dr. Kanthack that possibly the disease was sarcomatous. He (Mr. Shattock) took another view, regarding it as an inflammatory lesion, and the event proved that this was correct, for the patient died, many years after amputation, without any sign of recurrence. No doubt the condition must be closely allied anatomically to that produced by rickets upon bones. He had long taught that the thickening of the skull and long bones in rickets was a form of non-calcifying osteitis. These rhachitic changes must be classed among those due to infective processes. The lesions were not correctly designated "fibrous osteitis," since the nucleified tissue was not fibrous, but of a proper osteoid or bony kind, the deposition of earthy salts alone being deficient.

Dr. F. PARKES WEBER referred to a group of cases described in Germany under the heading of "fibrous ostitis" ("ostitis fibrosa of von Recklinghausen"), to which von Recklinghausen specially drew attention in 1891. During the last two years at least four papers had appeared on the subject.

Sarcoma appeared to supervene in these cases like it did in Paget's ostitis deformans. Dr. Weber suggested that the case so well described by Mr. Knaggs might be found to belong to the class of von Recklinghausen's fibrous ostitis ending in sarcoma, which, though in some respects allied to, was not identical with Paget's bone disease. A *localized* fibrous ostitis or "osteodystrophia cystica" had also been described by Mikulicz, Bockenheimer, and others.

Mr. KNAGGS, in reply, thanked the President for his replies to Dr. Parkes Weber's questions and points, as he did not himself profess to be a pathologist. He did not read German, and that was probably the reason he missed the papers to which Dr. Weber referred. He thought it was quite clear that his case was not one of ostitis deformans; there was nothing to suggest that condition in any way, either during life or post mortem. The nomenclature of the subject was somewhat difficult, and might be accountable for some of the confusion existing on the matter.

A Preliminary Note on Melanotic Deposits in Cattle and Sheep in Western Australia.

By J. BURTON CLELAND.

DURING the last year my attention has been drawn to the frequent occurrence in cattle and sheep in this State of deposits of melanin. Occasionally the original deposit was found in subcutaneous fibromata, but in many there was no tumour formation at all. I am at a loss to account for the presence of the pigment matter; a melanotic pigmentation of the livers and portal glands of sheep, which is very common at times, is supposed by the slaughtermen to be caused by the animals eating "saltbush" (order Salsolaceæ), but I have heard that this pathological condition occurs in parts of the country where the sheep cannot get this food. In two instances in bullocks there was an intense black deposit of melanin in the suprarenal glands, lying, however, not in the gland cells, but in granules in the supporting connective tissue cells, being especially dense in those of the vessel walls. In these two, also, there was a marked pigmentation, though not so deep as in the suprarenal gland, in the dura mater of the spinal cord, in wedge-shaped areas in the lungs and in the connective tissue around the aorta. Bearing in mind the marked pigmentation of Addison's disease, it is possible that in these instances, and perhaps in all the other cases too, the cortical cells of the suprarenal were responsible for the elaboration of the pigment,

which then in a soluble form passed on to the blood-vessels, in its course being stopped and "precipitated" by some of the connective tissue cells, and was from them taken up by cells in various other parts.

The cases so far met with are as follows:—

- (I) Deposit of melanin granules in the liver cells and portal glands of sheep. (Common.)
- (II) Deposit of melanin in the basement membrane of some of the renal tubules in a sheep.
- (III) Pigmented melanotic deposit in subcutaneous tissue of sheep, but without new growth.
- (IV) Melanotic areas in lung of sheep.
- (V) Intense black deposit in suprarenal gland, especially the cortex, of a bullock, with further deposits in the connective tissue cells of the dura mater, lungs, and tissue around the aorta. (Two marked cases.)
- (VI) Melanotic fibroma of neck of bullock with deep pigmentation of draining lymph gland, but without new growth therein.
- (VII) Intense melanotic deposit in a lymph gland near the hyoid bone of a bullock with discoloration of the surrounding parts.

(I) DEPOSIT OF MELANIN GRANULES IN THE LIVERS AND PORTAL GLANDS OF SHEEP.

These livers appear perfectly normal save for a greenish black colour, varying in degree, being often very dark and repulsive-looking. The greenish tint is due to the admixture of bile. On section, there is a beautiful marbling of the lobules, which show areolar black lines separating lighter portions. On first seeing the condition I paid little attention to it, supposing the dark coloration to be due to some bile-pigment. However, one day I noticed that the portal glands were, though not enlarged, of a dark slaty black colour; sections were therefore cut both of liver and glands. I may say that, though I was given to understand that these livers had a peculiar and rather pleasant taste, supposed to be due to the "saltbush," I could not detect any difference from the normal organ when cooked.

Microscopically, under a low power, the liver lobules were beautifully marked out by a dense deposit of melanin granules in the peripheral

zone. With a higher power these cells were found to be occupied by a great number of irregular small brownish black granules, especially heaped in the centre. The cells of the middle and inner zones were almost free from these granules, but many showed a rounded dark black mass in their centres. Scattered occasionally between the cells of the peripheral zone were large irregular cells densely packed with masses of pigment. Occasionally similar masses were seen in the connective tissue of the portal system. There was practically no free iron in the liver. The portal lymph glands were found to be deeply but irregularly pigmented. The pigment, which was in larger and more irregular masses than in the liver, appeared to occupy especially the cells of the stroma and to be most aggregated in the walls of blood-vessels.

(II) DEPOSIT OF A BLACK PIGMENTED SUBSTANCE IN THE BASEMENT
MEMBRANE OF RENAL TUBULES IN A SHEEP.

(III) BLACK PIGMENTED PATCH THE SIZE OF THE HAND IN THE
SUBCUTANEOUS TISSUES OF A SHEEP.

Microscopically, dense black pigment masses were found to lie between the bands of fibrous tissue.

(IV) LUNG, SHEEP.

Three or four *melanotic patches*, about $\frac{1}{4}$ in. long, on the pleural surface extending slightly into the substance.

(V) INTENSE MELANOTIC PIGMENTATION OF SUPRARENAL GLAND OF
BULLOCK WITH PIGMENTED AREAS IN LUNGS, DURA MATER, AND
AROUND THE AORTA.

Two cases of this condition occurred, exactly similar, save that one was more pronounced. In still another bullock, wedge-shaped black areas were found in the lungs, but unfortunately the suprarenal glands were not examined.

Suprarenal Glands.—In both animals the cortex of the gland was of an intense jet black, and on section exuded a dark brownish black fluid. The central part of the gland was conspicuous by its much lighter

colour. The connective tissue surrounding the gland was also pigmented with melanotic lines.

Lungs.—In both lungs several wedge-shaped areas 1 in. or more across were strikingly pigmented.

Dura Mater.—The dura mater of the spinal cord and base of the brain showed marked areas of bluish black pigmentation.

Connective Tissue around the Aorta.—This was streaked with lines of blackish pigmentation.

Microscopically, between the cortical cells of the suprarenal gland were dense irregular accumulations of brownish black pigment, varying in size from small granules to large masses. This pigment seemed to occupy and fill the connective tissue cells of the supporting stroma. In the lung the pigment, which was in large masses, again seemed to occupy the connective tissue cells. Its presence had given rise to some fibrosis and proliferation.

(VI) MELANOTIC FIBROMA OF NECK OF BULLOCK WITH MELANOTIC PIGMENTATION OF LEFT PAROTID AND SUBMAXILLARY LYMPH GLANDS.

Microscopically, in the lymph gland was found thickening of the fibrous septa, with extensive deposit of fine melanin granules in this near the parenchyma cells. There was an aggregation of pigment granules round elongated nuclei of connective tissue cells and, often in large amount, in the larger cells (connective tissue framework apparently) of the medullary spaces.

(VII) INTENSE MELANOTIC DEPOSIT IN A LYMPH GLAND NEAR THE HYOID BONE OF A BULLOCK AND DISCOLORATION BY PIGMENT OF THE SURROUNDING PARTS.

Microscopically, there was no evidence of new growth.

Pathological Section.

November 17, 1908.

Mr. S. G. SHATTOCK, President of the Section, in the Chair.

Case of Malignant Jaundice occurring during the course of Graves' Disease and associated with Gangrenous Tonsils.¹

By J. P. CANDLER.

THE following case, which I have termed acute biliary toxæmia, is, I think, worthy of record both on account of its own particular interest and because of its close resemblance, in many of the clinical and pathological features, to that variety of acute degeneration of the liver found in phosphorus poisoning and in the rare disease known as acute yellow atrophy.

The patient, a female, aged 38, single, and previously a housekeeper, was admitted into the London County Asylum at Claybury in September, 1901. She had been twice previously in this institution. She is described as suffering from recurrent melancholia. She was depressed, had suicidal tendencies, and was under the delusion that she was being followed about by people and that a gallows had been prepared for her because she had been the cause of the explosions in London. She also believed that her food was being poisoned; she complained at times that her head was bad. The thoracic and abdominal organs were normal, the tongue clean, and the teeth in good condition. There was no history or evidence of venereal disease. She was released from the asylum in March, 1904, but was readmitted after a month owing to a relapse; she continued in fair health until February, 1907. In September, 1907, she first exhibited symptoms of Graves' disease (exophthalmos, enlargement of the thyroid, irregular heart's action with tachycardia and tremors).

¹ From the Pathological Laboratory of the London County asylums.

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She became gradually worse, the thyroid enlargement increased, causing some difficulty in swallowing. On May 1 she had her first attack of vomiting, and by May 8 the condition had become so severe that nothing could be retained. She was given nutrient enemata and only sips of water by the mouth, but even this was returned. Her bowels were relaxed. She complained of great thirst and of pain and tenderness in the upper part of the abdomen. Jaundice was first noticed on May 4 as a slight icteric tinge of the conjunctiva. In the early morning of May 28 she sank into a state of complete unconsciousness and died in the afternoon of the same day, twenty-eight days after her first attack of vomiting and twenty-four days after the first appearance of jaundice. Shortly before her death she developed a purpuric rash. No increase or decrease was noticed in the liver dulness, and the cause of death was considered to be the exhaustion of Graves' disease. An autopsy was performed on the following day, the principal details of which are as follow:—

The bodily nutrition was poor, and the physique slightly below average. There was marked general jaundice, with a purpuric rash all over the body. The lungs were healthy; the heart was rather pale in colour, and the right chambers were dilated; there was no naked-eye evidence of fatty change, and no petechiæ. The aorta and great vessels showed evidence of early atheroma. Of the abdominal organs, the liver at once attracted attention. It was of a yellow ochre colour, with irregularly distributed reddish purple patches which were distinctly visible on the surface of the organ beneath a smooth and unwrinkled capsule. On removal from the body the organ was extremely friable; on section the purple areas were found scattered throughout the substance, which had a very granular appearance. The weight was 1,355 grm. (48 oz.). The gall-bladder contained greenish-coloured turbid bile; the mucous membrane was roughened and catarrhal. The spleen, soft and rather pulpy, weighed 120 grm. and appeared congested. The kidneys were enlarged (right 210 grm., left 180 grm.), soft, and swollen. The thickness of the cortex was increased; the cortex was pale in comparison with the deeper colour of the medulla. The capsule stripped readily, but left a slightly roughened surface on removal. Both suprarenal glands showed evidence of some disintegration. The uterus and appendages were normal. The bladder was contracted and empty, save for less than a drachm of rather turbid urine, which was preserved for examination. The brain showed nothing worthy of note. The thyroid showed a uniform enlargement; weight,

90 grm. There was one small cyst in the left lobe containing some glairy fluid.

Examination of the alimentary tract showed some features of considerable interest. The mucous membrane of the stomach and duodenum was congested and catarrhal, and this extended up the common bile-duct. The mucous membrane of the small intestine was also catarrhal, as was that of the large intestine to a less degree. The feature, however, of most interest was that both of the tonsils were in a state of almost complete disintegration. They were of an ashy grey colour, and in the case of the left practically only an outer shell remained, which enclosed a collection of fluid of slaty colour and of a most foul odour. The right tonsil still retained some vestige of its original shape, though from the crypts there could be expressed a fluid of a similar colour and of similar loathsome odour. They were practically gangrenous.

Cultures were taken from the liver, tonsils, and urine. In the case of the urine a small amount was obtained by thrusting a sterile pipette through the bladder wall, after the previous application of a cautery to the site of the puncture.

From the liver was isolated a Gram negative bacillus in pure culture, which, on further investigation, gave the characteristic reactions of the *Bacillus coli communis*. The organisms were not, however, present in sufficient numbers to allow of their being identified in a smear preparation made direct from some liver tissue and stained by Leishman's method.

From the tonsils the *Bacillus coli communis* was also isolated; but other organisms were present, the nature of which was not determined.

From the urine the *Bacillus coli communis* was isolated, but organisms of other species were also present. The fluid was albuminous and, microscopically, was found to contain bile-stained fatty tube-casts, individual fatty bile-stained epithelial cells, some red blood-cells, and a large amount of biliary pigment. Motile bacilli and other micro-organisms could be detected. Leucin and tyrosin could not be seen either in the urine or in the films of the liver, and a chemical examination of the liver extract met with a negative result.

MICROSCOPICAL APPEARANCES.

Liver.—The liver showed evidence of very marked and extensive changes in every part examined. The destruction did not appear to be limited to any particular zone, but was distributed throughout the liver

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lobule. The liver-cells had undergone extensive degeneration. Most of the cells which had not broken down were in a state of advanced fatty degeneration. In other places the cells were swollen, their nuclei were indistinct, and they appeared to be on the way to rapid disintegration. Distended capillaries and small hæmorrhages could be seen in places separating the degenerating liver-cells. Those cells which did not contain fat stained poorly, and in many instances could be seen to contain masses of bile-pigment, which was also present in large amount between the liver-cells. At the periphery of the lobule there was a

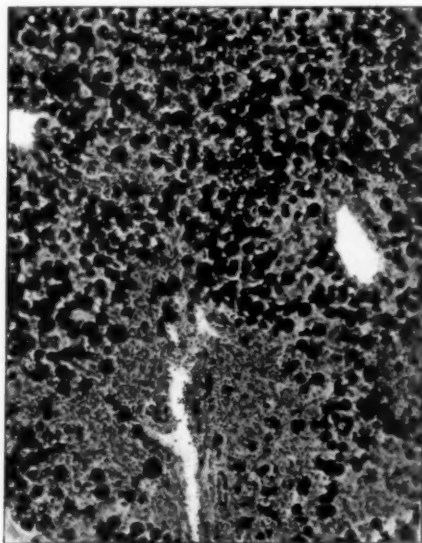


FIG. 1.
Liver (osmic acid). $\times 80$.

small-celled infiltration of the portal system and proliferation of the finer biliary canaliculi (fig. 1).

Kidneys.—The kidney changes were of the nature of a catarrhal nephritis. The cells of the convoluted tubules were mostly swollen; their nuclei were indistinct, and in places they had been discharged into the lumen of the tubules. Appropriate staining showed that their protoplasm contained numerous fat granules; the glomeruli appeared to have undergone very little change. A noticeable feature in the

specimens was the large amount of biliary pigment which could be seen, both in the lumen of the tubules and in the interstitial tissue (fig. 2).

Pancreas.—The pancreas showed some evidence of slight cloudy swelling, but otherwise there was little abnormal to be noticed.

Spleen.—The spleen showed considerable engorgement.

Duodenum.—The duodenum was the seat of a well-marked catarrh, especially of the mucous membrane. The capillaries in this region were distended, and the epithelial cells appeared to be undergoing a change of a necrotic nature.

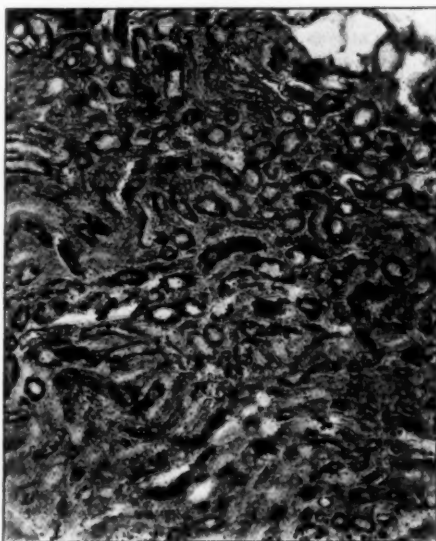


FIG. 2.
Kidney (osmic acid). $\times 80$.

Heart.—The heart showed no evidence microscopically of any fatty change, and presented a practically normal appearance except for some slight pigmentary infiltration of some of the muscle-fibres.

Thyroid.—The thyroid showed the changes usually found in cases of Graves' disease.

Brain.—A section of the brain tissue, stained by the Marchi method, showed the presence of fat granules in a few of the pyramidal cells of the cortex, and, further, a marked degeneration of the nerve-fibres of the white matter (fig. 3).

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From the above description I think that this case, which is undoubtedly one of malignant jaundice, bears a very close resemblance to, even if it be not identical with, the rare disease known as acute yellow atrophy. The close resemblance which exists between cases of phosphorus poisoning, malignant jaundice, and acute yellow atrophy, and more especially the very close resemblance between the latter two diseases, has been emphasized by Hunter.

The liver in the case I have described resembles that found in cases of phosphorus poisoning in one particular, *i.e.*, that there was no

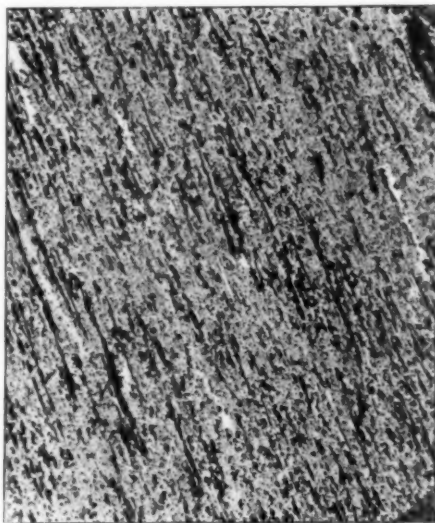


FIG. 3.

Brain; from white matter of frontal region. Marchi stain. $\times 80$.

apparent atrophy of the organ. In cases of phosphorus poisoning the liver, however, is usually enlarged and of a uniformly pale yellow colour, though in some cases patches of a reddish yellow colour may be found, due to congestion of the centres of the lobules. The organ is doughy to the touch and greasy on section. In this case the liver was mottled, and the yellow areas were of an intense orange or ochre colour; the organ was friable, and really had the feel of a liver which had reached the stage of advanced decomposition.

There are several points of resemblance in the clinical and pathological appearances to those found in acute yellow atrophy. The onset with vomiting, which later became uncontrollable, the appearance and gradual increase of the jaundice, the epigastric pain and discomfort, and the development in the later stages of a purpuric rash afford a picture closely comparable with that of acute yellow atrophy, but nevertheless common to cases of malignant jaundice.

The duration of the illness—four weeks, taken from the first attack of vomiting—is well within the range limit given by Thierfelder and Hunter, though the latter states that in the majority of cases it does not exceed fourteen days and rarely does it exceed three weeks. He further states that the duration of the first stage, which is made to terminate on the appearance of the nervous disturbances, varies from two days to three or four weeks. The average duration of the second stage in fatal cases is two to three days, though it may vary from twenty-four hours to fourteen days.

The case differs from the usual clinical picture of acute yellow atrophy in (1) the absence of well-established nervous phenomena, (2) the absence of retraction of the liver dulness and atrophy of that organ, (3) and the absence of leucin and tyrosin from the urine. With respect to the first point, I have ascertained that the course of the disease was unaccompanied by delirium, and the amount of restlessness noticed could be accounted for by the coexistent Graves' disease and the distress occasioned by the attacks of vomiting. The patient relapsed into a condition of complete unconsciousness on the morning of the day of her death. This may have been the precursor of the second stage, which at the most only lasted three or four hours, and might have been followed by the retraction of the liver dulness had the patient survived a few hours longer. In other words, I am suggesting, without asserting, that the coexistence of another grave disease was sufficient to bring about a fatal termination, either at the onset of the second stage before the liver had had time to retract or before the second stage had really developed. The absence of the diminution of the liver dulness during life and of shrinking and atrophy of the liver, as ascertained at autopsy, prevents this case from being called strictly acute yellow atrophy, for it is the sudden diminution in the volume of the liver which practically confirms the clinical diagnosis, which is further substantiated by the post-mortem findings. But there is ground for discussion as to whether a visible atrophy of the organ is a *sine qua non* in the diagnosis of this disease.

According to Opie, atrophy of the liver is not an essential feature of the disease, which, under the name "icterus gravis," is characterized by rapidly increasing jaundice, delirium, and other symptoms. In the early stages of this disease, according to Liebermeister, Orth, and other writers, the liver is enlarged rather than diminished in size. At this early stage the liver has not yet assumed the mottled yellow and red appearance, which is due to the presence of hæmorrhagic areas in which disintegration of the hepatic substance is far advanced; the tissue has an almost uniform bright yellow or ochre yellow colour. (I may state here that in the case under discussion the yellow areas of liver substance were in considerable excess over the purple areas.)

Hunter, on the other hand, considers that there is no support for this view. He states that "even in the bodies of patients who have died four days after being apparently in perfect health the liver has been found much reduced."

The atrophy of the liver is, in his opinion, one of the most characteristic features of the disease. In another place he states: "The diminution in the area of hepatic dulness usually does not become manifest until after the onset of the severe nervous symptoms, and often not till a few hours before death."

As regards the condition of the liver in the earlier stages of the disease (he adds), "observations are, unfortunately, but scanty, and for the most part those that exist are at variance. In the majority of cases the condition of the liver was not noted until the onset of the severe nervous symptoms, which directed attention to the true nature of the cases. By this time the diminution in size has actually begun. In a certain number of cases, however, in which earlier observations had been made, the stage of diminution was found to have been preceded by one of enlargement."

From the above observations it would therefore appear that the date of onset of this manifestation is not directly fixed to any particular time from the appearance of the initial symptoms, but is rather directly related to the commencement of what is termed "*the second phase*"; in other words, the onset of the nervous manifestations is in some way connected with the reduction in volume of the organ. Therefore, if a patient were to die either before the onset of the nervous phenomena or within three to four hours of their commencement, the inference would be that the liver might be found to be approximately of the normal weight and volume; and this is what I would suggest as a probability in this case.

Chemical examination of the liver of this case failed to detect leucin and tyrosin; but the presence of these substances, although furnishing, together with the other clinical and pathological features, a characteristic group of symptoms, is not essential to the diagnosis. Many instances of undoubted acute yellow atrophy have been recorded in which these substances have not been detected. Further, their presence in the urine has been recorded in cases of afebrile jaundice with slight enlargement of the liver, which bore no resemblance to acute yellow atrophy.

As to the precise etiology and nature of acute yellow atrophy no one is at present agreed. The opinion of the majority of writers on this subject is that the disease is due to the action of some poison which reaches the liver either by way of the arterial blood or from the alimentary canal by way of the portal vein. Those in favour of the latter theory point to the frequent association of catarrh of the gastrointestinal tract. The upholders of the theory that the poison reaches the liver by way of the general circulation draw attention to the presence of changes in other organs and consider that the excretion of the toxic agent by the bile would be sufficient to account for the pathological changes found in the bile-ducts and duodenum. So far as I can gather, the consensus of opinion seems to be that the poison enters by way of the alimentary tract. It might be expected that a microscopic investigation of the liver would furnish some evidence as to the initial site of the lesion in the liver lobule, but unfortunately the changes in the liver are so extensive at the time of death that this is rendered impossible. The evidence, however, that can be gained by examination of other organs of the body shows that, even if the lesion be localized in the liver in the first instance, the poison has spread to other organs by the time that death ensues. The disease is considered to be essentially a degeneration and necrosis resulting from toxic substances produced either by bacterial infection or through faulty cell metabolism—that is, by bacteriogenic or cytogenic toxins. Hunter considers that the widespread character of the liver changes and the rapidity with which they usually occur suggest the action of a circulating toxin rather than a local invasion of micro-organisms. The theory of the toxic origin is based mainly on the results which have followed from poisoning with toluylendiamin, phosphorus, and arseniuretted hydrogen.

As might be expected, micro-organisms have been cultivated from the liver and other organs. By some they have been considered the causative agent; others, more prudent, have acknowledged their presence but have refrained from assigning to them any specific characters.

Among the organisms so found are the *Bacillus coli*, organisms of the pyogenic group, especially streptococci, and a specific bacillus described by Guarnieri. Of these the *Bacillus coli* has been found more frequently than organisms of other species. It has been suggested that when organisms have been cultivated from the liver they have been present as a secondary infection, their growth being favoured by the lowered vitality of the tissues. My own observations lead me to believe that the examination of the various organs of the body post mortem is liable to lead to erroneous conclusions, so far as relates to the discovery of a specific organism, for as the result of a terminal invasion or immediately after death micro-organisms appear to multiply in the tissues at an enormous rate. In a recent investigation which I have made in connexion with the etiology of general paralysis of the insane, I have been much impressed by the number of organisms which can be obtained from film preparations of the organs, especially the brain, even in so short a time as one hour after death.

Attempts have also been made to assign to various disorders and conditions a greater or less share in the production of this disease. A very long list of such disorders can be quoted, including almost all the specific infective diseases, septic troubles, syphilis, alcohol, the disorders of pregnancy and parturition, chloroform poisoning, mental emotion, &c. Of particular interest in connexion with the gangrenous state of the tonsils described in this case is the description by Bertram Rogers of a case occurring in a child aged 4. The thymus gland was found enlarged and adherent to the apex of the right lung and pericardium. A cavity within it contained a small quantity of purulent debris, the remains of an old abscess. Particularly noteworthy is a case described by Cahn in a girl aged 17, in whom acute yellow atrophy accompanied diphtheria, affecting the tonsil and extending to the mucous membrane of the stomach. Bacterial inflammation of the stomach has been associated by Van Henkelon with acute yellow atrophy, and he has emphasized the probability that the hepatic lesion is caused by toxic material absorbed from the portal circulation.

With regard to experimental work, it is of interest to note that Boxmeyer has produced liver necroses by the inoculation of the bacilli of hog cholera. He induced two types of lesions, one of which was due to a plugging of the capillaries by endothelial cells, aided probably also by a direct action of the toxins upon the liver-cells. The other type could only be explained by the occlusion of capillaries by fused masses of red blood-corpuscles. He believed the fusion of these cells to be due to an

agglutinin. Pearce has claimed to have produced liver necroses by the venous injection of hæmagglutinative sera; he raises the question as to whether agglutinins may be a factor in the production of liver necroses in man. In attempting to bring agglutinative thrombi into relation with liver necroses in man it is necessary, he states, to explain the origin of the agglutinin. The possibility of its development as a bacterial product in the course of infectious diseases and toxic conditions naturally suggests itself. To those, therefore, who believe that acute yellow atrophy is due to the agency of bacteria and their products the suggestion offers itself that, by virtue of some devitalizing influence, such as some toxic or infectious disorder, bacteria of some definite species as yet undetermined, or even bacteria usually of a harmless nature, are aroused to activity, with the production of some form of toxin which, by its action, whether agglutinative or otherwise, is able to lead to destructive changes in the liver. Whether the agent which produces the atrophy, which is a characteristic feature of the disease, is specific and distinctive from that which is responsible for the changes in the liver which constitute malignant jaundice, or whether it is merely a question of degree of severity or some slight difference in the nature of the toxin, is a problem which still requires elucidation.

The case I have described is of interest by reason of (1) its association with exophthalmic goitre; (2) the presence of gangrenous tonsils; (3) the isolation of an organism possessing the characters of *Bacillus coli communis* from the liver and other organs.

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DISCUSSION.

Dr. F. PARKES WEBER said he understood from the author that there seemed a probability that acute atrophy of the liver was due to some specific cause common to all the various cases. He thought that there must be several different causes of acute hepatic atrophy. At the January meeting he intended to bring forward a case of acute hepatic atrophy occurring in secondary syphilis. There was almost certainly some causal connexion between the hepatic atrophy and the syphilis in such cases. P. F. Richter, in 1898, collected the records of forty-one cases of acute atrophy of the liver occurring in secondary syphilis, though he (Dr. Weber) had found that Dr. Richter had credited Hilton Fagge with two cases instead of one. Several other cases had been reported in the last ten years. All these cases of "icterus gravis" with acute atrophy of the liver in secondary syphilis were perhaps merely exaggerated forms of the cases of slight jaundice accompanying secondary syphilis. In favour of the jaundice accompanying secondary syphilis being causally connected with the syphilis, Arnheim observed, in at least two cases, that a patient who got jaundice with the first exanthem of syphilis again became jaundiced with a relapse of syphilitic sore throat. He (Dr. Weber) did not urge that Dr. Candler's present case was syphilitic in origin, though the mention of gangrenous tonsillitis just suggested such a possibility. But probably the author, on the other hand, did not wish to urge that the *Bacterium coli*, which had so often been discovered in the liver at post-mortem examinations in various diseases, was likely to have been the cause of

the hepatic atrophy in his patient. He would be glad to hear whether the patient had been questioned as to secondary syphilis. In the necropsies on cases of acute hepatic atrophy during secondary syphilis, spirochætæ had never been found in the liver, as they had been in cases of congenital syphilitic cirrhosis. The hepatic atrophy was certainly not caused by the local presence of spirochætæ in the liver, but might be due to their toxins. Most of the cases had occurred in females, and there might be something in the female organism which rendered their livers more vulnerable than those of men to the hypothetical syphilitic toxin. As evidence of syphilis in cases of acute hepatic atrophy, scarring (except from the primary chancre) could not be expected.

Dr. CANDLER replied that he did not suggest that the *Bacillus coli* was the cause of the disease, but like other observers he had found the organism in the liver post mortem, and had drawn attention to the fact. Every effort had been made to ascertain whether there was syphilis in the case, but there was neither history nor evidence of any kind. He had looked for any scarring on the mucous membranes which could be attributed to syphilis, and for any evidence of a healed primary sore. He recognized that it was impossible to state that any particular person had not had syphilis.¹ A number of cases had been described as occurring in young children, and in many there was no suggestion that syphilis was responsible for the condition. In this case there was some increase of connective tissue in the portal zone of the liver, but not of long standing. There was, however, a fairly well-marked cellular infiltration in the region of the peri-portal spaces, with slight proliferation of the bile-ducts.

¹ The suggestion that the condition of the tonsils might be due to secondary syphilis was negatived by the fact that the patient had been an inmate of the asylum for over four years.

The Mode of Action of Gastrotoxic Serum and the Healing of Gastrotoxic Ulcers.

By C. BOLTON.

THE serum referred to in this communication was prepared by immunizing the rabbit with the stomach-cells of the guinea-pig. The blood-serum of the rabbit during the process of immunization develops a poison for the guinea-pig's gastric cells, and also other poisons which produce slighter effects upon the other tissues of the body.

Two effects are observed as the result of intraperitoneal injection of this serum into the guinea-pig:—

(1) General symptoms of intoxication, which appear very soon after inoculation, death occurring within twenty-four hours if a fatal dose be administered.

(2) Post-mortem lesions limited to the stomach and consisting of blackened patches of necrosis in the mucous membrane, sharply defined and of variable size. Although usually multiple, one or two patches only may be found. The rest of the mucous membrane is normal. The necrosed tissue soon disappears, leaving sharply punched-out ulcers extending from the surface of the mucous membrane to variable depths, but not below the submucous tissue. Oedema of the subcutaneous tissue of the abdomen may be present, and very rarely a few hæmorrhages into the mucous membrane of the intestine.

MODE OF ACTION OF THE SERUM.

The stomach lesions may conceivably have been brought about in one of two ways:—

(1) By the direct action of the poison on the gastric cells, the hæmorrhage being secondary to disorganization of the mucous membrane.

(2) By a destructive action upon the capillary wall, the hæmorrhage being the primary condition.

That hæmorrhage is not the primary condition can be proved by the following experiment: About 20 c.c. of a solution of sodium bicarbonate of 1 per cent., or any strength above this, are introduced by œsophageal tube into the stomach of a guinea-pig. The animal is then inoculated

with a massive dose of gastrototoxic serum. At the same time another animal of the same weight is injected with the same dose of the same serum as a control. The animals will both die, and at the autopsy that in which the gastric juice had been previously neutralized will show absolutely no lesions in its stomach, but the control animal will show the usual patches of necrosis. Solutions of soda of strengths below 1 per cent. will diminish the amount of, but will not prevent, the necrosis. Neutralization of the gastric juice will not prevent a hæmorrhage occurring, and therefore it may be concluded that the poison acts primarily upon the gastric cells. But although this is the case, I have never been able to show that the serum actually causes necrosis of the gastric cells, because sections of stomachs in which the gastric juice has been neutralized show no microscopic lesion of the cells, such as is seen in the necrotic cases, although the animals have been killed by the serum. It appears, therefore, that a change is produced in the cells in some way or other whereby they are rendered susceptible to digestion by the gastric juice.

With regard to the nature of this pathological change in the cells, two hypotheses present themselves:—

(1) The cells possess a specific resisting power (possibly of the nature of an antibody) which is destroyed by the gastrot toxin.

(2) They are damaged or in some way devitalized.

That it is a question of devitalization and not the removal of some specific resisting power can be shown by testing the action of other protoplasmic poisons.

In a previous paper I was able to show that hepatotoxin, enterotoxin, and hæmolysin were able to produce necrotic patches in the stomach. Microscopically these patches are indistinguishable from those produced by gastrot toxin, but they are not so extensive and do not occur with such certainty. By the method of neutralization of the gastric juice it can be shown that these poisons act upon the gastric cells in precisely the same way as gastrot toxin, but the action is much more feeble. These poisons cannot be supposed to have any influence in removing any specific resisting power possessed by the cells, and yet they initiate self-digestion. It is therefore probable that the second hypothesis is correct, namely, that the cells are devitalized or damaged, and that the gastric juice digests them for this reason.

This observation is important from several points of view. It establishes the principle that self-digestion of the gastric mucous membrane may be brought about by a poison circulating in the blood, and it also raises the question whether there may not be many endogenous and also

exogenous poisons (*e.g.*, bacterial or otherwise) which, by their action on the gastric cells, may bring about this result.

The non-specificity of these sera seems to me of greater importance than if they acted specifically, because it extends the etiological field of acute gastric ulceration, which in the human being one finds associated with many different diseases in which a general poisoning of the system is the chief phenomenon. I showed two years ago that gastrotoxin contains many poisons. In the test tube it is hæmolytic and contains precipitins for the various tissues of the body; it especially acts as a poison for the gastric cells, but, as the symptoms and death of the injected animal show, it also acts as a general protoplasmic poison.

It is probably a fact that no cytotoxic serum is specific, but that each contains poisons which act upon most tissues of the body whilst affecting to the greatest extent the tissue against which the cytotoxin was formed. Hæmolysin is no exception to this rule; it acts upon blood-corpuscles, it acts upon capillary endothelium, and it possesses some action upon gastric cells. These experiments explain why so many observers have failed to obtain lesions on injection of cytotoxic sera, because a certain amount of damage may be inflicted upon a cell without any microscopic evidence of this being apparent. The stomach-cells are necrosed because they happen to be exposed to the action of a digestive ferment.

This question of the action of blood-poisons on the stomach-cells leads me to think that probably certain protoplasmic poisons introduced into the cavity of the stomach in solutions too weak to cause necrosis might, by devitalizing the cells, initiate self-digestion. I found such to be the case, and will now proceed to describe my results in this study.

EFFECTS OF HYPERACIDITY OF THE GASTRIC JUICE.

Hydrochloric acid is a well-known protoplasmic poison, and it seemed that possibly hyperacidity of the stomach contents might favour self-digestion. The acidity of the guinea-pig's stomach contents is about the same as it is in man. The animal is killed after eating a test meal and the total chlorides and inorganic chlorides estimated by the silver nitrate method. The following table shows the percentage of active HCl in several experiments at different intervals of time. The animals were fed on a normal diet and the stomach contents estimated each hour:—

Time after test meal			Percentage of active HCl
1 hour	0·1008, 0·126
2 hours	0·126, 0·108, 0·144, 0·144, 0·09
3 "	0·234, 0·216, 0·187
4 "	0·205, 0·252

As guinea-pigs are constantly eating, the percentage of HCl is probably about 0·1 to 0·2.

The effect of hyperacidity alone was first tested. The animals were first starved for a few hours and the solutions of HCl introduced by means of œsophageal tube. I found that strengths of HCl in distilled water up to 0·7 per cent. produced no effect whatever. Above 0·7 per cent. lesions occurred in the smaller animals and above 0·9 per cent. in the larger animals. The lesions produced were practically identical with those produced by gastrototoxic serum—namely, patches of necrosis and ulceration. Solutions above 1 per cent. strength converted the whole mucous membrane into a black mass.

The effect of hyperacidity combined with injection of gastrotxin was found to be a marked increase of the stomach lesions. Solutions of HCl which were innocuous alone (*i.e.*, below 0·7 per cent.) were introduced by œsophageal tube and gastrotoxic serum at once injected into the peritoneal cavity. Strengths below 0·25 per cent. to 0·3 per cent. (*i.e.*, below the normal acidity) did not increase the gastrotoxic lesions, but strengths between 0·3 per cent. and 0·7 per cent. all produced a marked increase.

It appears, then, that any strength of HCl above the normal can act as a protoplasmic poison for the gastric cells, and will add its quota to any other devitalizing influence and assist in bringing about self-digestion. I think this assistance to produce lesions of poisons which are in themselves innocuous is a very important point. The percentage of HCl in the human stomach rarely rises above 0·35 per cent. and never above 0·5 per cent., but the *total acidity* may rise to 0·6 per cent. or 0·7 per cent., so that it is probable that in the presence of some other devitalizing agent hyperacidity of the gastric contents may favour the production of ulcer.

That hyperacidity of the gastric juice increases the lesions produced by gastrotxin, by the HCl acting as a protoplasmic poison and not by increasing the peptic activity of the gastric juice, is rendered probable by the fact that only within small limits will an increase of HCl assist in the digestion of certain foodstuffs. Probably the whole question turns upon what strength of acid will most easily cause swelling of the tissue to be digested. This hypothesis is also supported by certain experiments in which I have introduced other protoplasmic poisons into the stomach.

Dilute sulphuric acid above 0·8 per cent., dilute lactic acid above 6 per cent., and dilute acetic acid above 2 per cent. will all give rise to necrotic patches in the mucous membrane of the stomach. In solutions

below these strengths the acids are inert, but all these inert solutions markedly increase the lesions produced by gastrotoxic serum in exactly the same way as HCl does.

I may here mention that vinegar as commonly used contains 4 per cent. glacial acetic acid, and it is by no means an uncommon practice for servant girls, who are liable to suffer from gastric ulcer, to consume large quantities of this substance. I have found that 0.5 per cent. acetic acid is capable of increasing gastrotoxic lesions. I have already stated that enterotoxin, hepatotoxin, and hæmolysin produce the same effects upon the stomach as gastrotoxin, but to a less extent. These effects may all be increased by the previous administration of inert solutions of HCl, H_2SO_4 , acetic and lactic acids; so that it appears that the action of all these cytotoxins is the same, the difference between them being one of degree only. I have further confirmed this point by testing the combining power of different tissues with gastrotoxin. The serum was exposed to the action of various cells of the guinea-pig previous to its injection. Care was taken to be sure that the cells saturated the serum in each case. The mixture was allowed to stand for one hour and was then centrifugalized. Two animals were injected in each experiment, one with the serum so treated and the other with untreated serum. I found that gastric cells were able in every case to completely remove the poison and render the serum inactive. Liver- and intestine-cells were able to do so to some extent. If the serum were a weak one the action might be destroyed, but otherwise it was reduced. Red blood-corpuscles were also able to some extent to remove the poison. All these tissues, therefore, show considerable combining power for the gastrotoxin; the latter is able to act upon each of these tissues *in vitro*, and sera formed against them will all produce some effect upon the stomach. Absolute specificity does not, therefore, exist, and I may add, without entering into the literature of the subject, that this result agrees with those of most workers on cytotoxins.

THE HEALING OF GASTROTOXIC ULCERS.

Since the gastrotoxin is a general protoplasmic poison, its injection into the peritoneal cavity is an unsuitable method to employ in order to study the healing of the ulcers. If a large dose which will certainly produce ulceration be given the animal will probably die, and if a small dose be employed one cannot be certain that ulceration will be produced,

because the poison is taken up by so many organs of the body that it is soon exhausted. I have therefore used the method of local injection of the serum into the wall of the stomach. By this method an ulcer can invariably be produced of a definite size in any position in the stomach without the occurrence of general toxic symptoms.

The guinea-pig in which an ulcer is to be produced is starved for about eighteen hours, so that the stomach wall is not too thin. The abdomen is opened in the middle line under strict antiseptic precautions and the stomach drawn out. A fine hypodermic needle connected with a glass syringe is inserted under the peritoneal coat of the stomach, and from $1\frac{1}{2}$ c.c. to 2 c.c. of the serum injected so as to form a blister under the peritoneal coat of the stomach. The size of this should not be less than that of a shilling. The stomach is replaced and the abdominal muscles and peritoneum united by silk, the skin being separately sutured. The wound heals by first intention. The serum soaks into the overlying mucous membrane, which is then digested at that spot by the gastric juice. That the action is toxic and not due to mechanical separation of the coats of the stomach, which might be supposed to cut off the blood-supply of the mucous membrane, can be proved by injecting neutral fluids in the same way. One may inject 2 c.c. normal rabbit's serum, normal guinea-pig's serum, or physiological salt solution, and the whole will be absorbed in twenty-four hours without leaving any trace of necrosis or ulceration. It is, of course, possible to produce slight mechanical ulceration by injecting enormous quantities of neutral fluids, but it is surprising to what an extent the mucous and muscular coats can be separated from the peritoneum without any harm whatever resulting. The activity of the gastrotoxin in producing an ulcer by this method varies exactly with its activity in producing lesions through the blood-stream. The best time for collecting the serum for this purpose is about six weeks after the commencement of immunization, and after about three months later the serum is not of much potency. The serum, then, produces digestion of the overlying mucous membrane, which forms a slough. There is always more or less hæmorrhage under the mucous membrane, which depends on mechanical injury to the vessels during the injection. By about the fifth day the necrosed portion has become disintegrated and separated, leaving a cleanly defined ulcer. The necrosed tissue undoubtedly becomes secondarily infected with bacteria from the food, and in this way adhesions with neighbouring organs and thickening of the base of the ulcer are produced. During the formation of the

ulcer the peritoneum may give way and general septic peritonitis ensue, or it may become adherent to the liver or other organ and a subdiaphragmatic abscess result.

The healing of the ulcers in normal animals has been studied in forty guinea-pigs. A certain number died during the formation from perforation; in the rest the ulcers commenced to heal at once, shrinking smaller and smaller until completely healed. Sometimes there is practically no thickening left, the ulcer having disappeared except for a slight puckering of the mucous membrane, which may have a stellate character; at other times there is considerable thickening of the peritoneum, and a definite nodule is left to mark the site of the ulcer. The base of the scar is often firmly adherent to a neighbouring structure, and loose completely organized adhesions may occur with the intestines, omentum, and abdominal wall. In all cases the ulcer heals completely in from fourteen to twenty-eight days. The actual time of healing depends on the size of the ulcer to a large extent and also upon the amount of inflammatory reaction, which in its turn probably depends upon the extent to which it has been secondarily invaded by bacteria.

I have no intention of discussing the literature on the subject in this short paper, but will merely remark that this result agrees with that of Cohnheim, who produced experimental ulcers years ago by injecting lead chromate into an artery so as to produce areas of infarction in the gastric mucous membrane, and it also agrees with the results obtained by many experimenters who directly injured the mucous membrane. In short, however produced, a gastric ulcer heals within a few weeks. I should like to draw attention to the similarity that exists between the ulcers on the table and many so-called chronic ulcers in the human subject. I believe that acute gastric ulcer is much more common than it is supposed to be, that in most cases it produces no symptoms except by accident, as for instance hæmorrhage, and that the majority of such ulcers heal up in a few weeks. I have notes of cases which illustrate these points. However, after making every allowance, one must admit that chronic gastric ulcer is by no means an uncommon malady, that it persists for long periods, and that it may extend. Since ulcers formed by the process of self-digestion are acute, there must be some condition or conditions which prevents the healing of such ulcers.

In an endeavour to throw some light upon this point I have performed the following experiments. (The following remarks, of course, apply to gastric ulcers in the guinea-pig.)

Repeated injection of the serum will not lead to chronic ulceration of the stomach as the animals develop immunity to the serum; 5 c.c. of inactive serum were injected each week, and after ten or twelve injections the animals were protected against a lethal dose of the serum. The serum of the immune animal also had the power of conferring passive immunity upon another guinea-pig, so that when it was injected with a mixture of gastrototoxic serum and immune serum no lesion resulted. *The position of the ulcer* does not affect the healing, as it is quite easy to produce an ulcer close to the *cardiac or pyloric orifice* or on the *anterior or posterior wall* of the stomach, and in all cases the ulcers heal up in the usual time in animals otherwise normal.

The effects of hyperacidity and diminished acidity of the gastric contents have also been studied. In order to test the effects of *hyperacidity*, ulcers were produced in animals, which were then fed on food soaked in 0.6 per cent. HCl. This is a high degree of acidity and the animals do not like it, but they will eat the food when they are hungry. The ulcers of animals on this diet healed up perfectly well, in fact, they healed rather more easily than in the control animals, probably because the food was better disinfected. The difference, however, was very slight. That it is possible to keep the acidity of the gastric contents above normal the following table will show. The animals were fed on food soaked in 0.5 per cent. HCl and the stomach contents estimated each hour.

Time after test meal			Percentage of HCl
1 hour	0.324
2 hours	0.2793
3 "	0.234
4 "	0.2916
5 "	0.3168

In order to test the effects of *diminished acidity*, animals in whom gastric ulcers had been produced were fed on food soaked in 4 per cent. sodium bicarbonate solution. The animals eat this food very well, and their ulcers heal up, but if anything not quite so well as in the controls, the average time of healing being slightly longer, probably because the food is not quite so well disinfected. It is not possible to keep the gastric contents permanently alkaline, as the following table shows. The animals were fed on food soaked in 4 per cent. sodium bicarbonate solution and the stomach contents estimated each hour:—

Time after test meal			Percentage of HCl
1 hour	Absent
2 hours	"
3 "	"
4 "	0.072
5 "	0.2016, 0.126, 0.252

The effect of feeding on alkaline diet is interesting, as it shows that the giving of alkali neither increases nor diminishes the secretion of HCl, each of which properties it has been supposed to possess. The HCl is neutralized by the alkali, till the latter is used up, and then the acidity of the gastric contents becomes normal; the average acidity in these cases is below normal, so that the food is not so well disinfected, and bacteria have a better chance of growing in the ulcer. It is impossible, therefore, to produce a chronic ulcer by a mere alteration in the acidity of the stomach contents.

DISCUSSION.

The PRESIDENT (Mr. Shattock) observed that the author had been at times credited with being able to explain the production of gastric ulcer in man by means of his work. The difficulty in any such affliction, however, was that in the experimental investigation the essential element was the action of a foreign serum. He asked Dr. Bolton to criticize a suggestion which occurred to him, viz., that in the case of chronic gastric ulceration in man the necrotic gastric cells furnished *in loco* a gastrotxin which lowered the vitality of the adjoining tissue or which was absorbed into the circulation, and so led to the extension of the lesion; and yet the same difficulty obtained here, for the toxin, being of autogenous source, would be inefficacious.

Dr. HERRINGHAM asked Dr. Bolton whether he thought gastric ulcers were more commonly present than diagnosed. Most people appeared to think they were less common, and that many cases of hæmatemesis were due to oozing from capillaries. This view had been supported on the ground that very few scars of old ulcers were found post mortem, but he agreed that this was not good evidence, since such scars were very hard to find. Yet there was hardly any other, for apart from the accident of perforation, cases of supposed gastric ulcer scarcely ever died.

Mr. BEDDOES asked what was the effect of giving insoluble substances? Allusion had been made to the practice among young girls of taking vinegar, but the practice of eating raw oatmeal was equally common, and the clinical result—anaemia—was the same. How did the oatmeal act?

Dr. BOLTON, in reply, said his investigation proved the principle that there might be a poison which would circulate in the blood and cause self-digestion, and certain substances, themselves innocuous, on introduction into the stomach would also, in the presence of some devitalizing condition in the blood, produce ulceration. He thought those facts would prove of importance in regard to human gastric ulcer. If one inoculated rabbit's stomach into a rabbit,

a poison was produced, but it did not act on the rabbit, though it did on the guinea-pig. Blood-serum of the normal rabbit was hæmolytic for guinea-pig's corpuscles; the hæmolysin was formed by absorption of the rabbit's own tissues. By absorbing the stomach of another rabbit it produced a poison which was toxic for nearly allied tissues. The same mechanism was concerned in producing an immune and an anti-immune body; therefore, if some condition could be found which would inhibit the formation of an anti-immune body while leaving the formation of an immune body intact, there would be auto-intoxication. But no one had yet been able to prove that. He did not think the President's suggestion concerning the debris of necrotic cells would work; first, because not sufficient was absorbed; and, secondly, whilst the animal was forming a poison against its cells it would immunize itself against that poison. He had been trying for a long time to produce chronic ulcers, and he thought some light would be thrown on that matter by some investigations he was making on motor insufficiency. The necrotic cells from the ulcer would be digested by the gastric juice, as in the case of any other foreign body, and form peptones. With regard to hæmatemesis resulting from capillary oozing, he quoted a case at University College Hospital, in which the patient vomited a considerable quantity of blood and died in a week, and clinically that case corresponded to the type of case in which there was said to be oozing rather than an ulcer; but by pinning the stomach out and carefully searching, an ulcer the size of half a threepenny piece was discovered, with a vessel passing over the floor of it. Much had been written on acute ulcers in association with infective disease, no doubt the result of bacterial poisons circulating in the blood. Sometimes the ulcers did not leave any scarring as in experimental ulcers. Many cases diagnosed as chronic gastric ulcer were not so; some diagnosed them from the mere presence of pain and vomiting. Hæmatemesis from capillary oozing formed a well-marked type. A few months ago he had a case of splenic anæmia which vomited a large quantity of blood, but there were only a few superficial erosions secondary to the hæmorrhage to be found. Hæmorrhage could occur by a poison acting on the capillary wall, or from the secondary opening up of vessels by acute or chronic ulcers. Probably infective diseases, such as yellow fever and hæmorrhagic smallpox and the purpuras and anæmias, were endothelial poisons. He did not think Hale White's gastrostaxis was as common as was thought by that gentleman, nor that it should be diagnosed without a post-mortem examination. He had never heard of raw oatmeal causing gastric ulcers. He thought raw oatmeal would act like chestnuts; a person who ate chestnuts had them collect in the rectum, and complained of constipation and inability to pass a motion, the anus being patulous from much straining and a discharge issuing therefrom; the finger on being inserted felt what seemed like a collection of sawdust, and when that was mechanically removed the condition was ameliorated.

The Action of Splenotoxic and Hæmolytic Sera on the Blood and Tissues.¹

By L. S. DUDGEON, P. N. PANTON, and E. ATHOLE ROSS.

THESE experiments were undertaken for the purpose of determining whether it would be possible to immunize an animal with splenic extracts so as to obtain a serum which would produce specific lesions in the spleen or lymphoid tissues generally, when injected into suitable animals, or if the general and specific effects observed could be referred to a hæmolytic action. The investigation was further extended to the action of these immune sera on the blood and tissues, with special reference to the production of fatty degeneration, cell necrosis, and the formation of "hæmolymp" glands.

TECHNIQUE.

The animals used in these experiments were rabbits, guinea-pigs, and cats. All the instruments—glass syringes, needles, vessels, and mortars—used were sterilized by boiling.

Preparation of Cytotoxic Serum.

A guinea-pig, after being killed under chloroform, was surgically cleansed and opened up over the splenic area. The spleen was removed, freed from fat, and pounded up in a mortar, with the addition of a sufficient quantity of citrated saline solution to make a good emulsion. This was filtered through sterile muslin. Various attempts were made to remove the red cells from the spleen without injury to the splenic cells, but the result was a failure. The splenic extract so obtained was then injected intraperitoneally (under aseptic precautions) into a rabbit. After the animal had received from seven to ten of these injections at intervals of about ten days, a further period of one week was allowed to elapse, and then it was bled from the carotid artery under an anæsthetic. The blood was allowed to stand in a sterile vessel for some time for the serum to separate out, and the process was further aided by centrifugation. The serum was next injected intraperitoneally into guinea-pigs,

¹ From the Pathological Laboratories of St. Thomas's Hospital.

and the results noted. The various doses used and certain modifications, such as the effect of heating the serum, will be subsequently referred to. As soon as possible after the death of a guinea-pig a complete post-mortem examination was made. Certain of the animals used recovered more or less completely, and these were killed and examined after a week or more, according to circumstances. A thorough macroscopic survey was first made, and then portions of the various viscera, together with the inguinal and mesenteric glands, were taken for microscopic examination. In certain instances the heart muscle and diaphragm were also examined. Paraffin sections were cut and then stained by hæmalum and eosin, Leishman and van Gieson's method. Staining for the free iron reaction was also undertaken, and gum sections were cut and treated with Scharlach R. for fat and with methyl violet for amyloid changes. Smears were made from the splenic surface, the bone marrow, and sometimes from the peritoneal surface and blood. These were stained with Leishman's stain and examined under the oil immersion lens, while in some instances differential counts were made. In a few cases the phagocytic power of normal washed human leucocytes was tested upon a 5 per cent. suspension of guinea-pig's red cells and of guinea-pig's spleen cells in the presence of normal, of immune, and of heated immune rabbit's serum.

Preparation of Hæmolytic Serum.

A serum hæmolytic for guinea-pig's red cells was prepared by injecting guinea-pig's red cells into rabbits. The blood was obtained direct from the heart or axillary artery, and collected in normal citrated saline solution.¹ From 8 c.c. to 10 c.c. of this citrated suspension of red cells were injected intraperitoneally into rabbits by a similar method to that employed in preparing cytotoxic serum. After the course of injections was completed, the rabbit's serum was drawn off from the carotid artery and used to inject guinea-pigs intraperitoneally. The results were noted, and complete autopsies were made on the pigs.

In the first two experiments the red cells were washed previous to injection in three changes of saline, but as this precaution did not affect the experiments in any way, it was discontinued. The important fact was thus demonstrated that a serum of equal hæmolytic potency was obtained whether the rabbit was injected with blood-cells and plasma or with blood-cells only. The rabbits furnishing the immune sera were in good health when killed.

¹ Sodium chloride, 0.85 grm. ; sodium citrate, 0.85 grm. ; sterile tap-water, 100 c.c.

Experiments on Cats.

Owing to the impossibility of obtaining splenic extract free from blood in the case of guinea-pigs, the following modified technique was devised with a series of cats in order to overcome the difficulty: The animal was anaesthetized and first bled from the carotid through a cannula into sterile citrated saline; a second cannula, previously inserted into the jugular vein, was then opened, and saline solution¹ at 37° C. was washed through the vascular system until the issuing stream was seen to be clear and almost free from colour. When the abdomen was finally opened up and the splenic vessels cut across, it was found that the saline gushed out at first reddish in colour, but that it soon became colourless and that all the viscera were remarkably pallid. Microscopic sections of the spleen were obtained, and proved this organ to be quite bloodless. The splenic extract was then prepared and used to inject a rabbit intraperitoneally. Another rabbit was at the same time injected with the cat's red blood-cells. These injections were repeated at regular intervals of ten days, and the general technique was the same as that previously given.

Finally, two series of cats were injected intraperitoneally with varying doses of the splenic cytotoxic and hæmolytic immune sera obtained from the treated rabbits. When the cats died complete autopsies were made, and the findings in the cats injected with splenic cytotoxic serum compared with the results in the other series inoculated with the hæmolytic serum. In certain instances the sera were heated before injection (twenty minutes at 60° C.) and other modifications introduced.

The post-mortem examinations included not only all the observations previously recorded when dealing with guinea-pigs, but also a chemical and spectroscopic examination of the urine.

Experiments in Vitro.

From time to time throughout this research the action of both the hæmolytic and cytotoxic immune sera was tested on a 5 per cent. suspension of washed guinea-pig's red cells and cat's red cells made up in normal saline. After the red cells had been obtained in citrated saline solution to prevent clotting, they were centrifuged and subjected to two further washings in normal saline solution. All excess of saline was

¹ 0.85 per cent. sodium chloride in tap-water.

pipetted off and then the red corpuscles were used in making the observations. The strength and dilutions of the immune sera used will be referred to later. A constant volume of red cells was placed in a set of hæmolytic tubes and then the immune serum was added diluted with normal saline in varying proportions. The tubes were sealed up, incubated for two hours at 37° C., and subsequently placed in the ice chest overnight. An examination of the tubes was made next morning and the amount of hæmolysis noted. Similar experiments were also conducted with heated serum, and control tests were employed with normal serum.

Serum was subjected to the direct action of moist heat in the following way: The serum tube, after being sealed up, was placed in the bottom of a test tube containing sufficient water at 58° C. to cover three-fifths of the tube. The test tube itself was then transferred to the well of an oven filled with water kept to the temperature of 58° C. by a gas regulator. The heating was continued for twenty minutes or more, and, in certain instances, a temperature of 60° C. was used and the heating carried out in a precisely similar manner.

Examination of the Rabbits' Blood.

In the earlier portion of this research the blood of the rabbits was examined during the time the injections were given with a view to the elimination of sepsis. The results were noted in the case of four rabbits injected with guinea-pig's splenic extract and of one injected with guinea-pig's red cells. The examinations were made immediately before a fresh injection.

An analysis of from forty to fifty blood-examinations thus made showed that in the rabbits undergoing inoculation there was never a large leucocytosis, and in the differential count the polymorphonuclear cell was never the predominant one at the end of the treatment. In other words, the blood-examination did not suggest the presence of complications of an acute inflammatory nature.

In every instance the rabbits and cats when killed were healthy, except for the presence of a little peritoneal thickening and a few adhesions at the site of inoculation. Therefore the immune sera used in our experiments were obtained from animals in a sound condition, and the success of the precautions against infection during the inoculations was proved. In an investigation like the present the importance of eliminating sepsis cannot be over-estimated.

GENERAL RESULTS.

The General Results which Followed the Injection of the Sera.

Under this heading are considered the more immediate and clinical effects which followed the administration of cytotoxic and hæmolytic sera. In addition, the broad results are noted of those experiments in which either the sera were heated or else treated with red cells in the hope of removing the hæmolysin, and of the few cases in which animals of one species were inoculated with sera immunized for animals of a different species.

(a) *General Effects of Cytotoxic Sera.*—These sera were obtained from rabbits which had been treated with the spleens of guinea-pigs or cats in the manner previously described. Seven rabbits were injected with the spleens of guinea-pigs and one with the spleens of cats. The cytotoxic serum so prepared was used to inoculate twenty-four guinea-pigs and three cats. The sera of the various animals thus immunized showed some individual differences in potency. In the case of one rabbit which had received nine injections, lasting over a period of three months, a serum was obtained of somewhat higher toxic powers than the sera from the other rabbits; $\frac{1}{2}$ c.c. of this serum killed a guinea-pig in a few hours, whereas a guinea-pig recovered after receiving 5 c.c. of the serum of another rabbit. On the whole, however, the effects produced by the serum from the different rabbits were fairly constant, and death of the guinea-pig, as a rule, resulted when the dose exceeded 1 c.c. In each series of guinea-pigs the fatal effect coincided in time with the amount injected, and death took place in from a few hours to three days or more. The immediate result of the injection was, in most instances, a condition of severe shock; a partial recovery then frequently took place, to be followed later by relapse and death. In those experiments in which cats were injected small doses of serum in comparison with the body-weight of the animal proved to be fatal. These animals died in from two to three days, and showed marked symptoms from the time of inoculation; they became listless, dyspnoic, their conjunctivæ assumed a distinctly yellowish tinge, and they developed hæmaturia. In those experiments in which an animal had been injected with the spleens of a second animal, and its serum used to inoculate another species of animal, no toxic effects were obtained; thus a pig received 5 c.c. of the serum of a rabbit which had been inoculated with the spleens of cats and remained perfectly well; whereas a cat on receiving the

same dose of the same serum was dead in twenty-four hours. In another example a cat received 5 c.c. of the serum of a rabbit immunized with guinea-pig splenic emulsion without any untoward effect, while a pig was killed in a few hours by a similar dose of this serum. The effects of injecting heated serum were contrasted with those of unheated serum in the case of a few animals, and it was found that the serum thus decomplemented was slower in its action and had less immediate toxic effect, but did not differ materially in its final result. For example, a guinea-pig was given 2 c.c. of heated immune rabbit's serum and died in thirty-six hours; three pigs in the same series which received $\frac{1}{2}$ c.c., 1 c.c., and $2\frac{1}{2}$ c.c. unheated serum died in twelve, five, and two hours respectively. A few attempts were made to eliminate the hæmolytic action of the cytotoxic sera. In one experiment the serum was saturated with red cells *in vitro*,¹ but the general effects produced by it were in no way diminished. In two other instances guinea-pig's red cells were introduced into the peritoneal cavity of a pig immediately before the injection of the immune serum. The results of this proceeding were to avoid the preliminary shock and to prolong life. To cite an instance, a guinea-pig was inoculated with the red cells of a normal guinea-pig and immediately afterwards with $\frac{1}{2}$ c.c. of immune serum; it died in twenty-four hours. A second pig received $\frac{1}{2}$ c.c. of the same serum only and was dead in five hours. It will be seen that these results are comparable with those obtained by decomplementing the serum by heating it.

(b) *General Effects of Hæmolytic Sera.*—These sera were obtained from rabbits which had been treated with the blood of guinea-pigs or cats in the manner described above. Three rabbits were injected with the blood of guinea-pigs and one with the blood of cats. Ten guinea-pigs and three cats received injections of the sera of these rabbits. The serum obtained from one rabbit which had been given nine injections, lasting over a period of three and a half months, was slightly more toxic than the sera of the other rabbits; doses of 1 c.c. and 3 c.c. killed guinea-pigs in two hours. The hæmolytic sera as a whole appeared to be rather more deadly than the cytotoxic sera, the injections of the former being invariably fatal, but in no instance was a dose less than 1 c.c. given, whereas in the case of the cytotoxic sera very small doses, such as $\frac{1}{10}$ c.c. and $\frac{1}{20}$ c.c., were occasionally used; however, recovery exceptionally took place after as much as 5 c.c. of cytotoxic serum. Death followed

¹ This serum was not proved to have entirely lost its hæmolytic property before inoculation.

the injection of the hæmolytic sera in from two to forty hours. In the experiments in which cats were injected a train of symptoms was set up precisely similar to that obtained with the cytotoxic sera; the onset of the symptoms, however, was more acute, and death took place more rapidly. In one experiment 1 c.c. of the serum of a rabbit which had been immunized with the blood of a cat was injected into the peritoneal cavity of a guinea-pig and no ill-effect followed; a similar dose given to a cat killed it in twenty-eight hours. The result of this experiment is analogous to that obtained in a similar series of experiments on cytotoxic sera. The object of these tests was to demonstrate the specific nature of the immune serum employed. The effects of injecting heated hæmolytic sera were contrasted with those of unheated sera in a few instances. The difference was found to consist in a diminution of the initial shock and in a postponement of death. For example, 8 c.c. of the heated hæmolytic serum of a rabbit killed a guinea-pig in eighteen hours, whilst doses of 3 c.c. and 10 c.c. of unheated serum caused almost immediate death; in the case of another rabbit 3 c.c. of serum killed a guinea-pig within twelve hours, while with a dose of 4 c.c. of heated serum life was prolonged for twenty-four hours. In two instances attempts were made to eliminate the hæmolytic action of these sera. In one case the serum was saturated with guinea-pig's red cells *in vitro*, and no alteration whatever in the toxic properties of the serum for the guinea-pig was produced. In another case a guinea-pig received an intraperitoneal inoculation of guinea-pig's red cells, immediately followed by $\frac{1}{2}$ c.c. of hæmolytic serum; the animal recovered. Two other pigs were given 1 c.c. and 3 c.c. of the hæmolytic serum only; both died in two hours. The saturation of the serum with red cells *in vitro* appeared to have no effect on its subsequent action; when, however, the serum was permitted to act upon the red cells in the peritoneal cavity of the animal a marked difference in its toxicity was obtained. The animal in question was the only one to recover of all those which received hæmolytic serum.

Summary.

So far as the general results of injecting these sera are concerned the ultimate effect appears to be the same whether a cytotoxic or a hæmolytic serum is employed. On the whole, the hæmolytic serum was more rapid in its action than the cytotoxic. The act of heating the sera, whether cytotoxic or hæmolytic, merely postponed death. In the case of the cytotoxic sera attempts to remove the hæmolytic action had no

more effect than that obtained by heating the serum. In the one example in which the hæmolytic serum was saturated with red cells *in vivo* the general action of the serum was successfully neutralized.

DETAILED RESULTS FOLLOWING THE INJECTION OF THE SERA.

(I) *Macroscopical Post-mortem Changes.*

(a) *Following the Injection of Cytotoxic Sera.*—The post-mortem changes varied considerably with the dose of serum employed and the time which supervened before death. In those animals which recovered and were subsequently chloroformed practically no naked-eye changes were observed. The most characteristic appearances were seen in the animals which died in a few hours and after a large dose; in these cases an excess of fluid was often present in the pleural and peritoneal sacs, and in a considerable proportion of the guinea-pigs numerous small hæmorrhages had occurred, chiefly into the peritoneum and mesentery, but also into the pleural surfaces of the lungs, the pericardium, the visceral surfaces, into the muscles, and rarely in the stomach. The most striking changes were seen in the spleens. These were greatly swollen, softened, and congested; their colour varied from purple to almost a jet black, causing them to stand out in marked contrast to the remaining viscera, which were, as a rule, abnormally pallid. In the majority of post-mortems, including those on the animals which had recovered, a few red or dark-coloured glands, of an appearance suggesting hæmolymph glands, were found in the groin, while the mesenteric glands were often considerably enlarged and sometimes dark in colour. In one case the small intestine was deeply congested, and in another case it was filled with a deeply blood-stained fluid. The appearances in the cats were even more pronounced: the fat was of vivid primrose yellow colour, and the viscera and conjunctivæ had a marked "icteroid" tinge; the liver and kidneys were extremely pale, the spleen swollen and blue black. Blood-stained urine was present in the bladder, but there were no hæmorrhages. When the serum was heated before injection, changes resulted similar to those which followed the unheated serum.

(b) *Following the Injection of Hæmolytic Sera.*—The changes found at the post-mortems in this series of animals were very similar to those present in the animals which had received cytotoxic sera. An excess of peritoneal fluid was usually present, hæmorrhages were particularly abundant, and, in the case of two pigs which died within two hours, the small intestines were filled with a deeply blood-stained fluid. Enlarged

and red lymphatic glands were numerous, but the spleen-changes were not so constant as in the cytotoxic cases, and in some rapidly fatal cases the spleens appeared normal to the naked eye; in those cats, however, which received hæmolytic sera the spleens were as large and as black, while the other changes were practically identical with those observed in the cats which had been given a cytotoxic serum. No difference resulted from the heating of the hæmolytic sera nor from previously saturating the sera with red cells *in vitro*. The one pig which was given one injection of pig's red cells, followed by $\frac{1}{2}$ c.c. of hæmolytic serum, recovered and no naked-eye post-mortem changes were found other than a few somewhat enlarged and reddish lymphatic glands. In three separate instances we found that serum obtained from an animal of species A, by immunizing it against the splenic extracts or red cells of one of another species B, was innocuous when inoculated into a third animal of species C, and nothing abnormal was to be seen post mortem in the latter. In one pig, however, which had been given 5 c.c. of the serum of a rabbit immunized with the spleens of cats, a few diaphragmatic hæmorrhages were present, but the spleen and other viscera showed nothing abnormal.

(II) *Histology of the Organs.*

A histological examination was made of the spleen invariably and of all the more important viscera in the majority of instances, and it was frequently found that marked pathological changes were present in viscera which appeared perfectly normal to the naked eye.

The Spleen.

(a) *After Injecting Cytotoxic Sera.*—In the most typical sections the splenic sinuses were so widely distended and so filled with red blood-corpuscles and endothelial cells as to render the tissue almost unrecognizable as that of the spleen; in the Malpighian corpuscles, however, the changes were much less pronounced. One of the most striking features in the specimens was the extreme amount of phagocytosis which had taken place, the large endothelial cells being packed with red blood-corpuscles and golden brown pigment. The endothelial cells in some instances contained much more pigment than red cells. In the Malpighian corpuscles the endothelial cells not infrequently contained small lymphocytes and occasionally red blood-corpuscles—as a rule the phagocytosis in this situation was slight; here also fragmentation of nuclei was frequently well shown and mitotic figures were present. In

the sinuses granular debris was common, agglutinated masses of red cells were met with, and in other cases the red cells were hæmolyzed. In some instances these changes were much less marked, and very occasionally, when very small doses had been given, little abnormal was seen in the sections. In those experiments in which the serum was heated before injection or in which pig's red cells were added, similar changes were found in the spleen. The iron reaction, although always tested for, was generally poorly shown and occasionally absent; the golden brown pigment above referred to, which constituted a marked feature in many of the sections, did not give the true iron reaction. In the large majority of instances smears were made from the splenic juice and the degree of phagocytosis was noted. The following are typical examples: One hundred endothelial cells were counted, of which twelve were phagocytic and had engulfed nineteen red cells; in another instance a similar number of red cells were counted—fifteen were phagocytic and twenty-six red cells were engulfed. The phagocytosis thus observed in the smears was considerably less than appeared in an examination of the sections.

(b) *After Injecting Hæmolytic Serum.*—A careful comparison of the splenic sections from the animals in this series with those of the cytotoxic series failed to discover any points of distinction. Changes precisely similar in degree and kind were effected in the spleens whether a splenotoxic or hæmolytic serum was injected, and it may be recalled that in the case of the cats every precaution was taken to render the spleens bloodless.

Histology of Glands.

(a) *Cytotoxic.*—The lymphatic glands were examined in the majority of cases, and were taken from the mesentery and from the groin. The mesenteric glands as a rule were considerably enlarged, and sometimes mottled with dark areas; the glands of the groin more frequently appeared normal, but in some instances the naked-eye appearance was suggestive of hæmolymph glands. The most typical changes were found in the enlarged mesenteric glands; the glands of the groin, however, were occasionally similarly affected. Sections of the glands in typical cases showed under the low power considerable areas in which the *lymphoid cells* had *disappeared*¹ and the gland trabeculæ stood out, lying among a tissue of loose texture; the germinal centres were as a rule poorly defined, the general appearance of the section resembling

¹ This change was possibly due to the action of the immune sera.

a partially wasted spleen rather than a lymphatic gland; subcapsular hæmorrhages were not infrequent. The open spaces under the high power were seen to consist of sinuses containing red cells, endothelial cells, scanty lymphocytes, blood debris, and golden brown pigment; the most striking feature of the sections was the extreme degree of phagocytosis present in these sinuses; numbers of the endothelial cells were distended with engulfed red blood-corpuscles, which in some cases were so closely packed within the cell that it was impossible to enumerate them; less frequently the endothelial cells contained the pigment.



FIG. 1.

Section of the spleen of a cat which died in fifty-six hours after an intraperitoneal injection of 5 c.c. of splenotoxic serum. Section shows replacement of the lymphoid tissue by large areas of blood and golden brown pigment. Large cells containing red blood-corpuscles seen throughout the section. (Eye-piece B, objective 3.)

Phagocytosis was proceeding also in the lymphoid nodules, and here the cell most frequently engulfed was the lymphocyte. Polynuclear neutrophile cells as a rule were extremely scanty; in one case, however—namely, in the mesenteric gland of a pig which had received 2 c.c. of heated cytotoxic serum and died in thirty-six hours—these cells were

very numerous in the sinuses, and the endothelial cells were actively phagocytic towards them; red cells in this case were almost absent from the sinuses. In some of the glands numerous definite capillaries filled with red blood-corpuscles were found. Fragmentation of the nuclei of the mononuclear cells was a conspicuous feature in a few of the sections. A free iron reaction was only exceptionally obtained, and this reaction was not given by the golden brown pigment. Smears made from the glands and stained by Leishman's stain showed little abnormal, the greatly predominating cell being the small mononuclear,



FIG. 2.

Section of spleen of a guinea-pig which died in eighteen hours after an intra-peritoneal injection of 5 c.c. of splenotoxic serum. Marked distension of the splenic sinuses with blood, and these also contain numerous large mononuclear cells packed with red blood-corpuscles. Very little lymphoid tissue remains except in the Malpighian corpuscles. (Eyepiece B, objective $\frac{3}{8}$.)

the granular cells and the endothelial cells being proportionately scarce; nucleated red cells were usually absent; very little evidence of phagocytosis was found in the smears. While these changes in the glands have been described as typical, and were present to a greater or less extent in the majority of the glands examined, all gradations were

observed, and in a few of the glands but little or nothing abnormal was noted. The extent to which these changes had advanced did not seem to have been affected by the dose of the serum given, by the heating of the serum, by the attempts to remove the hæmolytic action of the serum, nor by the periods which elapsed between the inoculation and the death of the animal.

(b) *Hæmolytic*.—The glands¹ of the animals which had received hæmolytic sera were examined in the same way, and precisely similar changes were found.



FIG. 3.

Section of mesenteric gland from the same guinea-pig as in fig. 2. There is replacement of the lymphoid tissue by large sinuses distended with blood-corpuscles and containing endothelial cells packed with erythrocytes.

The Kidney.

Very marked and fairly constant changes were present in these organs. The most obvious effects were produced upon the convoluted tubules; in this portion of the kidney the tubular epithelium was swollen, partially or completely necrosed, and the seat of extreme fatty

¹ This change was possibly due to the action of the immune sera.

degeneration; the lumina of the tubules was either obliterated by the swollen epithelial cells or packed with red blood-corpuscles. These changes of advanced necrosis and fatty degeneration were practically confined to the convoluted tubules, and as the straight tubules were reached the appearances changed abruptly—no fatty degeneration was present, the epithelium showed merely varying degrees of cloudy swelling, and only scattered red cells were present in the lumina. No casts were seen in the tubules. The fatty change in the kidneys was more marked and more thoroughly examined in the case of the cats. In

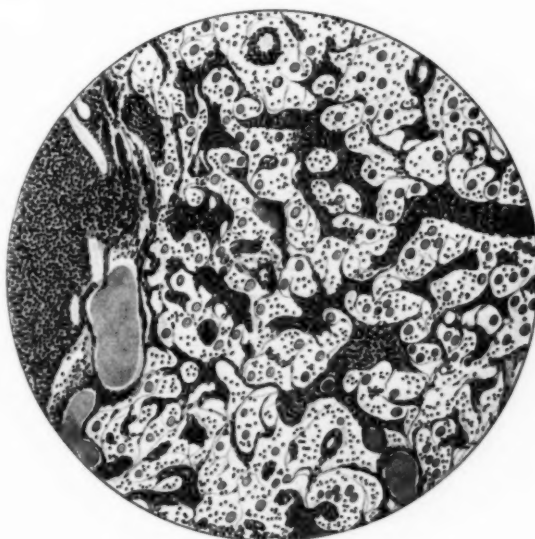


FIG. 4.

Section of mesenteric gland of a guinea-pig which died in five hours after an intraperitoneal injection of $\frac{1}{2}$ c.c. of cytotoxic serum. Section shows replacement of the lymphoid tissue to a large extent by spaces containing endothelial cells and scattered lymphoid cells. (Eyepiece B, objective $\frac{3}{8}$.)

these cats it was found to be extremely advanced in those animals which died earliest, namely, in from twenty-eight to forty hours, and appeared before any fatty change had occurred in the liver; in those animals which died after a longer period, namely, after fifty-six and seventy-six hours, advanced fatty degeneration was now present in the liver. The glomeruli were often filled with blood, and the epithelium of

Bowman's capsules was sometimes shed, but no fatty change was present. Areas of hæmorrhage and isolated red blood-corpuscles were scattered throughout the connective tissue of the kidney, and particularly in the cortical portion. Golden brown pigment was frequently present in the epithelial cells; a free iron reaction was usually absent.

In nearly every instance the urine in the bladders of these cats contained a large amount of albumin, and was a deep red colour owing to the presence of blood; no casts were seen, and the deposit contained blood debris only. Clumps of spermatozoa were almost constantly present. The spectrum was that of oxyhæmoglobin and the urine contained no bile.



FIG. 5.

Section of liver of the same cat as referred to in fig. 1. Section shows widespread areas of cell necrosis; towards the central vein large numbers of "vacuoles" are present in the necrosed cells.

The Liver.

The leading characteristic of most of the sections was the widespread areas of cell necrosis. Usually this was more marked in the centre of the lobules than at the periphery; in some of the sections it was best shown at the peritoneal surface; this might be due to the direct action

of the serum on the liver. The hepatic cells showed all gradations, from complete necrosis to mere swelling and indifferent staining reaction. In some of the sections a large amount of pigment was present, especially along the course of the capillaries. Phagocytosis of red cells by endothelial cells was noted in some of the specimens. Fatty change was often well marked, particularly around the central veins, and in some instances had proceeded to an advanced degree. While fatty degeneration and cell necrosis were frequently present in the same section, the two processes appeared to be independent, and necrosis was more constant and often advanced when fatty change was absent. A free iron reaction was often present, but never intense. No bile-staining of this or any other viscus was observed. No amyloid change

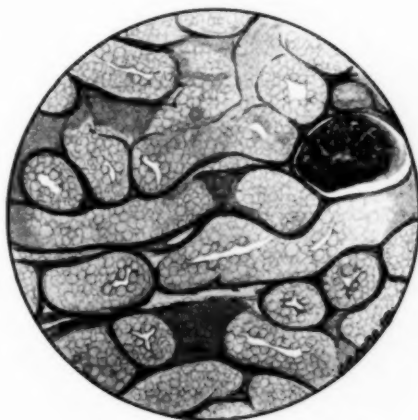


FIG. 6.

Section of kidney of cat which had received 1 c.c. of hæmolytic serum and died in twenty-eight hours. Section shows necrosis of the epithelium of the convoluted tubules, which is also considerably swollen, causing almost entire obliteration of the lumen. The necrosed epithelium contains innumerable "vacuoles." (Eyepiece B, objective 3. Stained with hæmalum and van Gieson.)

was detected. The liver and kidney changes were sometimes extremely marked in those animals which recovered from the effects of the injection, or from many small injections, and were subsequently killed, widespread areas of necrosis and marked fatty change being present in the liver.



FIG. 7.

Section of the liver of a cat which had received $2\frac{1}{2}$ c.c. of *heated* splenotoxic serum intraperitoneally and died in seventy-six hours. Section shows widespread fatty degeneration throughout the hepatic lobules, least marked towards the periphery. (Eyepiece B, objective $\frac{3}{4}$. Stained with Scharlach R. and hæmalum.)



FIG. 8.

Section of kidney of a cat which had received an intraperitoneal injection of 1 c.c. of hæmolytic serum and died in twenty-eight hours. Section shows widespread fatty degeneration of the convoluted tubules, the glomeruli remaining intact. (Eyepiece B, objective $\frac{3}{4}$. Stained with Scharlach R. and hæmalum.)

Other Viscera.

The heart muscle and diaphragm were normal except in the case of one cat, where a slight fatty change was noted. The pancreas and adrenal glands frequently showed some areas of necrosis. The intestinal epithelium showed no fatty change, but the villi were occasionally necrosed. The blood-vessels throughout the viscera were as a rule engorged with blood.

The Blood and Bone-marrow.

No bile was present in the serum, and the spectrum was that of oxyhæmoglobin in the cases examined. A few blood-films were stained for fat with a negative result. The bone-marrow was mainly composed of non-granular cells, the small mononuclears predominating. In a few instances the red cells were broken up to an extreme degree, the fragmented cells forming a granular background to the film; there was no alteration in the number of nucleated red cells. Phagocytosis of the red cells was slight in the marrow films, and no phagocytosis of the mononuclears was noted. The blood gave the spectrum of oxyhæmoglobin in every case. The serum was golden yellow in colour, but contained no bile and did not show a blood-spectrum.

IN VITRO.

Experiments with Immune Sera in Vitro.

In nearly every case experiments were conducted *in vitro* for the purpose of gauging the hæmolytic power of the immune sera. The test was always made on a 5 per cent. suspension of guinea-pig's or cat's red corpuscles in 0.85 per cent. of sodium chloride. The method of making these experiments has already been sufficiently indicated. The serum was diluted in the usual manner for conducting *in vitro* experiments, and the highest dilution used was 0.12 per cent. of immune serum.

The Cytotoxic Immune Serum.

The sera were obtained by injecting rabbit's with guinea-pig's splenic extract. As is well known, the serum of a normal rabbit when added to the red cells of a guinea-pig produces hæmolysis. In order to obtain a

set of control experiments the hæmolytic action of normal rabbit's serum on guinea-pig's red cells was tested.

Synopsis of Results with Immune Serum.

In the case of the first rabbit the experiment was performed after the fifth inoculation. Hæmolysis was complete with an immune serum content of 12·6 per cent. and very slight with one of 0·25 per cent. One of the guinea-pigs which had received a dose of 2 c.c. of this immune serum recovered, and this animal's serum was tested against guinea-pig's red cells. No hæmolysis occurred.

In the case of the second rabbit the experiment was performed after the fifth inoculation. Hæmolysis was complete with an immune serum content of 25·3 per cent., nearly complete with one of 12·6 per cent., and a slight action occurred in the presence of 0·12 per cent. of immune serum.

In the case of the third rabbit the experiment was performed just before the seventh inoculation. Hæmolysis was complete with an immune serum content of 2·5 per cent. and incomplete down to the last dilution of 0·12 per cent. When this serum was heated in a sealed tube at 58° C. for twenty-five minutes the hæmolytic action was destroyed.

In the case of the fourth rabbit the experiment was performed immediately after the sixth inoculation. Hæmolysis was complete in the presence of 2·5 per cent. of immune serum and present, though slight, down to a dilution of 0·12 per cent. When this serum was heated for twenty minutes at 58° C. a very slight reaction was found with an immune serum content of 50·6 per cent.

The experiment was repeated after the final inoculation, with the following result: Hæmolysis was complete in the presence of 2·5 per cent. of immune serum and incomplete in that of 0·12 per cent. The action was this time decidedly more powerful. When this serum was heated for twenty-five minutes at 58° C. the hæmolytic action was destroyed.

In the case of the fifth rabbit the experiment was performed after the fourth inoculation. Hæmolysis was complete with an immune serum content of 12·6 per cent. and very slight in a dilution of 0·12 per cent. of serum. The hæmolytic action of this serum on human red cells was only partial, and absent in the presence of 38 per cent. of immune serum.

In the case of the sixth rabbit the experiment was performed after the fourth inoculation. Hæmolysis was practically complete with an immune serum content of 12·6 per cent. and just present with a dilution of 0·12 per cent. of serum. When this serum was heated to 60° C. for twenty minutes all the hæmolytic action was destroyed. The immune serum obtained after death from this rabbit was also tested against guinea-pig's red cells. The result agreed with the previous experiment, except that hæmolysis was absent in the highest dilution of 0·12 per cent. of immune serum.

Finally, one rabbit received injections of cat's splenic emulsion which had been washed free from red cells, and after the fourth inoculation this animal's serum was tested on a 5 per cent. suspension of cat's red cells. Hæmolysis was complete with an immune serum content of 25·3 per cent., partial with that of 2·5 per cent. and present with 1·26 per cent. of serum. Moist heat at 60° C. for half an hour destroyed all hæmolytic action of the immune serum. As a control the action of normal rabbit's serum on cat's red cells was tried. Hæmolysis was very slight with an immune serum content of 12·6 per cent.

The Hæmolytic Immune Sera.

The sera were obtained by treating rabbits with intraperitoneal inoculations of guinea-pig's or cat's red cells. The hæmolytic experiments with these sera were performed in the same way as before, and their action tested on guinea-pig's and cat's red corpuscles.

Synopsis of Results with Immune Serum.

In the case of the first rabbit the experiment was performed after the third injection. Hæmolysis was complete with an immune serum content of 2·5 per cent. and slight with that of 0·12 per cent.

In the case of the second rabbit the experiment was performed after the fifth inoculation. Hæmolysis was complete in the presence of 12·5 per cent. of immune serum and incomplete, but marked, in that of 0·12 per cent. Heating to a temperature of 58° C. for twenty minutes destroyed hæmolytic action. On repeating the experiment after the eighth inoculation the immune serum was found to have slightly increased power. Hæmolysis was then nearly complete with an immune serum content of 2·5 per cent. and rather more marked than

before in all the higher dilutions. Heat applied for twenty minutes at 58° C. again sufficed to destroy the hæmolytic action. In this case we reactivated the heated immune serum with guinea-pig's complement and repeated experiments reported in which we used rabbit's complement. In each instance hæmolytic action was restored, but was not quite so powerful.

In the case of the third rabbit the experiment was performed after the fourth inoculation. Hæmolysis was complete in the presence of 12·6 per cent. of immune serum and very marked in a dilution of 1·26 per cent. With the highest dilution used (0·12 per cent.) it was slight but distinct. As a result of applying moist heat for fifteen minutes at 58° C. hæmolytic action was not destroyed till the immune serum content was reduced to below 50·6 per cent.

One rabbit was treated with injections of cat's red corpuscles (5 per cent. suspension in 0·85 per cent. sodium chloride solution, as used in all instances). The action of this immune serum was then tested on cat's red cells and the experiment was performed after the eighth inoculation. Hæmolytic action was complete with an immune serum content of 12·6 per cent. and present in the highest dilution of 0·12 per cent. Moist heat acting for thirty minutes at 60° C. destroyed all hæmolytic action.

The Cytotoxic and Hæmolytic Immune Sera Compared.

Attention has already been directed to the greatly increased hæmolytic action of all the immune rabbits' sera on guinea-pigs' red cells as compared with that of untreated rabbits' serum on the same cells. Both cytotoxic and hæmolytic immune sera produced hæmolysis in the higher dilutions of 2·5 per cent. to 0·12 per cent. It was only the fact that the hæmolytic immune sera produced a greater degree of hæmolysis in these high dilutions that proved them rather the more potent.

The important point which these experiments *in vitro* have brought out is that if we immunize a rabbit against guinea-pig's splenic cells, *i.e.*, if we endeavour to produce a specific splenic cytotoxin for guinea-pigs, we obtain a serum which is powerfully hæmolytic for guinea-pig's red cells *in vitro*.

It will be remembered that the cats' spleens were washed free from blood as far as possible previous to being pounded up for inoculation into the immune rabbit. Yet, nevertheless, we obtained a "rabbit-cat"

splenic cytotoxic serum which was powerfully hæmolytic for cats' red cells.¹

When we further allow for this observation having been made early in the course of the immunizing process it cannot be argued that the "rabbit-guinea-pig" splenic cytotoxic sera always owed their hæmolytic action to the red cells unavoidably injected along with the splenic emulsion; hence, whatever specific action such a cytotoxic serum might possess, there was no doubt that it was powerfully hæmolytic—a fact which had to be reckoned with from the outset.

PHAGOCYTOSIS.

Just a few experiments were made with a view of testing the phagocytic power of normal washed human leucocytes on guinea-pig's red blood-corpuscles in the presence of immunized rabbit's serum. It was found, both in the case of the hæmolytic and of the cytotoxic immune serum, that phagocytosis was increased towards guinea-pig's red cells even when the serum was diluted as much as 1 part in 20. In control experiments with normal rabbit's serum phagocytosis was practically absent. In no instance were small mononuclear cells observed to be included by the polymorphonuclear cells in a similar set of experiments, when the red blood-corpuscles were replaced by an emulsion of guinea-pig's splenic cells. In all these experiments, although the hæmolytic power of the immune sera was notable, at the same time phagocytic action was quite slight in comparison.

CONCLUSIONS.

(1) There is no evidence that the immune serum obtained by injecting splenic extracts into animals possessed any specific action either upon the spleen or upon the lymphoid tissues in general.

(2) The action of the splenotoxic serum was practically identical with the action of the hæmolytic serum.

(3) The injection of splenic extracts, freed from blood, so far as it is possible to do so, yielded immune sera of undiminished hæmolytic potency.

(4) Animals injected with either washed or unwashed blood-cells gave sera of identical properties.

¹ Normal rabbit's serum produced only very slight hæmolysis of the cat's red cells when the content amounted to 12·6 per cent.

(5) Experiments conducted *in vitro* showed that the splenotoxic and hæmolytic sera had almost equal power to hæmolyse red blood-corpuscles.

(6) Heated splenotoxic and hæmolytic sera produced effects similar to those obtained with the untreated sera.

(7) The main effects upon the viscera of injecting these immune sera were :—

(a) To produce widespread necrosis of epithelial cells, particularly in the liver and kidney.

(b) To bring about an extreme degree of fatty degeneration in the same viscera and, unlike diphtheritic toxin, to spare the heart muscle and diaphragm.

(c) To greatly distend the sinuses of the spleen with red blood-corpuscles at the expense of the lymphoid tissue, leaving the Malpighian corpuscles comparatively unaltered, and to induce in the endothelial cells a high degree of phagocytosis for erythrocytes and blood-pigment.

(d) To cause the formation of areas in the lymph-glands of the mesentery and groin of similar histological appearance to the changes observed in the spleen, that is to say, a production of the so-called "hæmolymph" glands.

DISCUSSION.

The PRESIDENT (Mr. Shattock) remarked that some of the questions raised by the contribution were very embarrassing. Since the hæmolytic serum was not specific, on how many tissues did it positively act and produce necrosis? It looked as though the red blood-cell was in reality a highly complex structure, or rather, perhaps, that it held in the meshes of its stroma a complex fluid in which all the constituents of the body were represented, as if it were a carrier of fluid containing extracts of all the body-cells. Another curious point which the experiments brought out was the resistance of the Malpighian bodies to the action of the splenotoxic serum. Lymphocytes were particularly hardy cells. Some years ago, when he was working with Mr. Ballance on the subject of cancer, they used to place small pieces of recently excised carcinomata into test tubes of serum, and allow them to remain for long periods, at the end of which the fragments were examined by means of microscopic sections. In the sections of such macerated (sterile) tissue, although the general cells failed to take

any stains, the nuclei of the lymphocytes stained so well that at one time they thought they were dealing with living parasites which had survived in the necrotic tissue.

Dr. BOLTON asked whether Mr. Dudgeon said that when he saturated hæmolytic serum with blood-corpuscles and injected it into animals, the results were the same as when untreated serum was injected. Also, did they test *in vitro* to see if the serum was hæmolytic? In his own experiments he had found that all the tissues of the body would remove the hæmolytic amboceptor except the stomach, and on injecting the gastrot toxin treated with stomach-cells into the guinea-pig it had no effect. It was a surprise to him that the blood could not be washed out from the spleen by introducing a cannula into the thoracic aorta, since the same could be done via the jugular vein.

Dr. PARKES WEBER asked whether there was real jaundice present in the animals experimented on. He suspected that in some cases of chronic (slight) acholuric jaundice in human beings with splenomegaly it was not invariably possible to demonstrate bile-pigment in the blood-plasma.

Is there an "*Idiopathic Dilatation*" of the Urinary Bladder?

By S. G. SHATTOCK.

THE above question was forced upon me by a very unusual specimen which was sent to the Royal College of Surgeons by Mr. William Angus, the Resident Medical Officer of the Chelsea Infirmary, and which is now in the College Museum. The specimen consists of a much-dilated and hypertrophied bladder, with associated double hydronephrosis and dilatation of the ureters. The remarkable feature in the case is that there was nowhere any recognizable source of obstruction and that the patient had never experienced any difficulty in micturition.

The term "*idiopathic*" I use in this connexion in the manner in which it is used in the case of the colon and œsophagus, as implying a dilatation with hypertrophy, without tangible or obvious cause. The designation "*neuropathic*" is, of course, more correct, for we can hardly doubt that such conditions arise from defects in the innervation of the viscus concerned; but vesical dilatations result from very different nervous causes, and to name it in this way would not draw attention to its peculiarities. I do not propose to engage in a desultory discussion upon neuropathic dilatations of the bladder in general, for they comprise a variety which includes that arising in hysteria, that following operations upon the rectum and anus, or for varicocele, or the radical cure of hernia, or amputation of the lower limb, and operations upon the uterus and ovaries; that, again, which is due to injuries of the spinal cord or brain; or which is met with in the course of tabes. The so-called atony of the bladder which follows over-distension of the healthy organ is probably also essentially neuropathic in its pathology, and results from damage sustained by the nerve-plexus upon and within the muscular wall rather than from the stretching of the muscle-fibres themselves.

Before, however, describing the specimen in detail I may refer briefly to the kindred subject of idiopathic or neuropathic dilatation and hypertrophy of the œsophagus and of the colon, and to the views of their pathogenesis which offer themselves for consideration.

IDIOPATHIC DILATATION OF THE ŒSOPHAGUS.

Most museums contain one or more preparations of this condition, and they are all so remarkably alike that the citation of one or two will

suffice. In the Museum of St. Thomas's Hospital there are two; the first of these (Specimen No. 912) is an extremely dilated œsophagus. The dilatation increases from the upper end to the junction of the middle and lower thirds, where the canal measures about $5\frac{1}{2}$ in. in circumference; beyond this it diminishes until, at the cardiac orifice, the œsophagus regains its normal dimensions. The mucous membrane is superficially ulcerated for considerable areas; the muscular coat, though stretched, is as thick as natural, from hypertrophy. The stomach was of the ordinary size and perfectly healthy, and its cardiac and pyloric orifices in every way normal. [From a woman, aged 60, who died of peritonitis from perforating ulcer of the duodenum. There was tubercular disease of the liver and bronchial glands. The ulceration of the mucosa in this case is exceptional, and was probably secondary in pathogenesis.] The second specimen (No. 912 A) is the lower 8 in. of an œsophagus with the cardiac end of the stomach. The œsophagus is throughout dilated (measuring where most so 2 in. in diameter), but without any obvious cause. A thick piece of glass rod has been passed through the cardia. The muscular wall of the dilated canal presents no signs of atrophy. [From a man who suffered for thirteen years from œsophageal obstruction, and who ultimately died of inanition.] But one of the most pronounced examples is that in the Museum of University College Hospital, from a patient who was under the care of Dr. Sidney Martin (Specimen No. 1522). The œsophagus is so greatly dilated that, in its central part, it has a circumference of 12 cm. At the upper and lower ends the dilatation gradually diminishes, the upper end of the œsophagus and the cardiac orifice being both normal in size. To the naked eye the muscular coat presents no change. [From a woman, aged 35, admitted in September, 1892. The duration of the illness was two years; it commenced with vomiting and pain in the epigastrium; attacks of dyspnœa followed, and towards the end there was cough with muco-purulent expectoration. At the autopsy the dilated œsophagus was found filled with undigested food; it pressed on the pericardium and bulged into the right pleura; there was no stricture. There was broncho-pneumonia with recent pleurisy at the base of the right lung.]

To recall the physiological data in regard to the œsophagus. The tube is innervated by the vagi. The conduction of its peristaltic wave does not depend, as it does in the case of the intestine, upon an intramuscular nerve-plexus (Auerbach's), so that if the tube is transversely divided the nerve-supply around it being left intact, peristalsis, if excited

in the upper segment, is still transmitted to the lower. The nervous impulse which induces the peristalsis also brings about an active dilatation of the cardiac sphincter. Although a dual impulse is conducted by the same nerve, it is, of course, by different fibres, a physiological fact of which there are many other examples.

(1) It is conceivable in the first place, then, that a neuropathic dilatation might arise from a paralytic condition of the vagi. This would involve a passive distension of the canal with ingested food driven into it by the pharyngeal constrictors, the cardiac sphincter remaining at the same time unrelaxed. Involuntary muscle, however, will contract quite apart from a nervous impulse, as a result of mere tension or stretching. This is demonstrated by the simple experiment of filling an excised piece of artery under pressure. As there are no ganglia in the arterial wall, the possibility of a local reflex may be excluded, and the contraction which ensues must be attributed solely to the stimulus induced by the tension of the muscle-fibre itself. Hence, such a paralytic tube would, upon the distension reaching a certain grade, be excited to contract for the above reason, and although in an imperfect degree, yet sufficiently to force some of its surplus contents through the sphincter into the stomach. Against this view (which may exceptionally be correct), however, is the fact that the muscular wall of the dilated tube is not thinned or degenerate, although the disease may have lasted many years. The same anatomical evidence against a simple paralytic distension holds in idiopathic dilatation of the colon, where we find the muscular wall in no way thinned, but even thicker than normal. And the same is notably so in the case of idiopathic dilatation of the bladder which forms the subject of the present communication. One has, of course, in all these instances to differentiate the atrophy and fatty change in the muscle arising as a secondary or terminal condition from the disease itself, as in the case of the final cardiac dilatation which may succeed a compensatory hypertrophy.

(2) A second possibility is that there is an obstruction arising from incoördination between the action of the expellent musculature and the cardiac sphincter, the latter failing to relax simultaneously with the contraction of the tube above. This hypothesis would account for the slowly increasing dilatation and the absence of muscular atrophy—it accords with the anatomical results actually witnessed.

There are two further possibilities:—

(3) That the excitation of the peristaltic reflex is not the mechanical distension of the tube, but the stimulation of the mucosa. This will

appear from the circumstance that quite an insignificant amount, or minimum, of material will excite the action and be followed by its transit into the stomach. An under-sensitive or hypo-æsthetic state of the mucosa might theoretically, therefore, be followed by a diminished reflex and by a chronic accumulation of ingesta, neither the tube being excited to contract nor the sphincter to dilate.

And, lastly, (4) a contrary condition affecting the cardia, a mucosal hyperæsthesia, might be the cause of a local contraction of the cardiac sphincter as often as its passage was attempted, the result being an obstructive dilatation and hypertrophy of the tube above. In speaking of hypo-æsthesia and hyperæsthesia of a mucous membrane I do not, of course, limit the application of the terms to conscious, tactile sensibility.

DILATATION AND HYPERTROPHY OF THE STOMACH ASSOCIATED WITH HYPERTROPHIC STENOSIS OF THE PYLORUS.

In the explanation of this condition the two views which most commend themselves for consideration are: (1) Incoördination between the expulsive action of the stomach and the relaxation of the pylorus; and (2) spastic contraction of the pyloric canal arising from a hyper-æsthetic condition of the pyloric mucosa—an hysterical pylorus; the latter is the view to which I incline. On this hypothesis the attempted passage of the normal contents would of itself excite a reflex closure. It receives some support by analogy from the phenomenon of enterospasm. In this disease a particular segment of intestine is prone to contract, and so tightly as to produce obstinate attacks of obstruction or constipation, without any trace of organic disease, the subjects of this condition being more particularly of the neurotic class. The spasm, as Dr. H. P. Hawkins¹ remarks, may be short, acute, and intensely painful; or, with less violence, it may be of much longer duration, and may result in a steady ache or discomfort which will last off and on for weeks or months at a time. The colon is more commonly affected than the small intestine. Of the colon any part may enter upon the spastic state, but the first and last parts are particularly liable to be so affected.

And to the same group of phenomena belong those cases in which, without any evidence of organic disease, the ingestion of even easily digested food is followed by pain and sometimes vomiting. "This

¹ *Brit. Med. Journ.*, 1906, i, p. 65.

hyperæsthetic state or nervous dyspepsia may develop without any previous illness."¹

"Associated with hyperacidity there is often a real gastric hyperæsthesia, either a primary sensory neurosis or a secondary result of continued hyperacidity, and the mere contact of food with the gastric mucosa will then excite pain and discomfort."²

In the urinary organs such cases are paralleled by those in which (in women especially) a persistently increased frequency of micturition, perhaps hourly, takes place without any recognizable cause.

IDIOPATHIC DILATATION OF THE COLON.

As contrasted with the œsophagus, the peristalsis of the small and large intestine is conducted by the intramuscular plexus of Auerbach; and physiological experiment has shown that after transverse section of the gut the peristalsis ceases to pass from the upper to the lower segment; it has to be restarted independently in the gut below by the insertion of a second bolus. After end-to-end union of the divided intestine, the upper segment undergoes a certain degree of dilatation from this cause; there is a block or break in the peristaltic impulse, and to that extent a certain amount of impediment, although the peristalsis of the upper segment, by forcing contents through the functionless zone, will lead to the reinduction of peristalsis in the segment below. In the case of the ureter, since no nerves are demonstrable in the muscular wall, the peristalsis is supposed to arise from the direct contact of muscle-cell with muscle-cell; but no such conduction obtains in the intestine. In the heart, too, the contractile impulse is conducted by direct muscular continuity, the wave of contraction from auricle to ventricle being transmitted by the auriculo-ventricular bundle, division or lesion of which is followed by block and incoördinate action. Nothing of this kind can be applied to the dilatation of the pelvic colon which commonly arises in infancy and persists throughout life. No anatomical break in the muscular continuity can be here alleged, and if anything of the nature of a block obtains in this condition, it would be a nervous and not a muscular one. That the obstruction is in some instances spastic appears from direct observation—from digital examination *per rectum*—for the rectum has been found in a state of spasm.

¹ H. P. Hawkins, "Diet in Disease of the Stomach," in "A System of Diet and Dietetics." Edited by G. A. Sutherland (1908).

² H. P. Hawkins, *loc. cit.*

contraction; and in other cases the anus, the rectum itself participating in the dilatation of the colon. This pathogenesis, nevertheless, does not exclude the possibility of other explanations in other cases. The constancy of the site at which the dilatation arises has led to the theory of its being due to a folding or kink of the gut, seeing that in nearly all cases the disease involves the lower 3 in. of the pelvic colon, at the spot where the mobile bowel begins to lose its mobility and its mesentery together, and merges into the rectum. Let me only suggest, then, that the mechanical kinking is the starting-point of the disease, in that it may, by the compression and damage done to the intramuscular nervous plexus, lead to a functional block, the interruption in the peristalsis being followed by the slow dilatation of the colon above. And, finally, there is the possibility that the mucosa is hypo-aesthetic, whence there comes about a want of response to normal stimuli, the reflex being delayed until the colon has become filled to an unnatural degree.

IDIOPATHIC DILATATION OF THE BLADDER.

I may now describe the specimen which suggests the occurrence of an idiopathic dilatation and hypertrophy of the urinary bladder, of the same kind as that met with in other viscera, and adduce evidence furnished, also, by further specimens and observations, in support of such a supposition.

The bladder, which was sent to the Royal College of Surgeons in March, 1908, was taken from a man who died in the Chelsea Infirmary. It is enlarged so as to measure 5 in. in diameter. Its muscular wall is nevertheless well developed throughout, and measures 0.5 cm. in thickness. On the interior the bundles of the innermost layer of muscle, which have more or less of a vertical disposition, stand prominently out, and beneath them the thickened bundles of the circular series. The prostate gland is not in the least enlarged; its macroscopic structure is spongy and natural; nor is there any trace of an abnormal uvula or other mechanical source of obstruction. It may be observed that the vesical cavity does not shelve off into the prostatic portion of the urethra, but is bounded by an abrupt, well-defined edge at the site of the internal meatus. Both the ureters are greatly dilated, but their apertures are of normal size and preserve their proper obliquity. I evacuated the urine in the bladder and the chief hydronephrotic kidney; it was quite clear, of a pale yellow colour, and not ammoniacal.

Histological Examination.

Sections cut from portions of the wall, hardened in alcohol and stained with hæmatoxylin and eosin, display stout bundles of normal muscle-cells, some in cross section, some in longitudinal. Sections made from other portions, which had been preserved in dilute alcohol, were stained for twenty-four hours in Scharlach solution after being first passed through 75 per cent. alcohol; these were, after staining, washed in 75 per cent. alcohol, then in tap-water, and finally mounted in Farrant's medium. One of the sections was stained with hæmatoxylin before being placed in Scharlach. In the sections thus tested for fat no trace was found in the muscle-cells.

The patient was a cabdriver and a heavy drinker. On a previous admission to the Chelsea Infirmary he was suffering from profuse hæmorrhages from the bowel, such as occur in leukæmia, and an examination of the blood showed the marks of this disease. He presented no indications of tabes; he did not suffer from hæmorrhoids. For the last twenty-four hours of life there was incontinence with overflow, and 65 oz. of urine were withdrawn ten hours before death; the bladder again filled.

The catheter used (No. 7) passed without difficulty. The urine contained a large amount of albumin. There was no history of any renal colic or of any difficulty of micturition. Mr. Angus was good enough to critically examine the urethra at my request, after the bladder had been forwarded to the College, for it was sent only as a good example of dilated and hypertrophied bladder, combined with great dilatation of the ureters and double hydronephrosis. He informs me that he found no signs of obstruction in the urethra, which allowed a full-sized bougie to pass quite easily, and on slitting it up there were no signs of old disease. The heart and liver were somewhat fatty, and there were considerable adhesions in both pleural cavities.

Such conditions as hypertrophic stenosis of the pylorus (with the ensuing dilatation and hypertrophy of the stomach), and idiopathic dilatation with hypertrophy of the pelvic colon, certainly occur in infantile life, the neuro-muscular defect commencing to manifest itself very soon or immediately after birth. To establish an analogous condition in the urinary bladder, it will therefore strengthen the case if the same incidence of age can be brought out, and this I will endeavour to do.

Dr. Dawson Williams has recorded in the *Transactions of the Pathological Society* (xxxix, p. 152) the case of a male child, aged 5½ years,

who died of uræmia. He had "wetted the bed every few minutes" for nine months, but had never complained of any pain or difficulty in micturition. About six months before he came to the hospital it was noticed that the lower part of his belly was swollen, and that he was passing an unusually large quantity of water; since that date the swelling of the abdomen had constantly increased, and he suffered much from thirst. There was no phimosis, and a catheter was passed without difficulty, but the stream only flowed when pressure was made on the abdomen. He was passing large quantities of pale urine which contained a trace of albumin. After death it was found that pressure on the greatly distended bladder caused regurgitation into the ureters; urine readily flowed by the urethra. The ureters and renal pelves were much dilated; both kidneys were fibrotic. The child's mother stated that he had always been delicate, and had been suffering from diarrhœa for several months.

Dr. John Thomson has recorded the case of a male infant, aged 17 days, who presented enormous dilatation of both ureters, with very great hypertrophy and dilatation of the bladder, and no discoverable obstruction in the course of the urethra.¹

The reason why such dilatations of the œsophagus, the stomach, or pelvic colon are not met with as congenital lesions arises from the obvious circumstance that the organs in question do not functionate until after birth. With the urinary bladder it is different. The bladder receives and evacuates urine as a normal phenomenon during intra-uterine life. This is proved by the distension which occurs in cases of imperforation of the urethra, of which there are many examples on record. But what if a similar distension is met with without urethral malformation? Let me cite two instances in which this is so. One of these is in the Museum of the Royal College of Surgeons (Specimen No. 4724 c). It is the trunk of a seven months female foetus, the abdomen of which has been laid open to display a greatly dilated urinary bladder. The consequent distension of the peritoneal cavity produced dystocia, and during delivery the bladder, together with the abdominal wall, was ruptured. The mother, aged 25, had been in labour many hours when first seen, and the head was already born. The bladder was large enough to contain 3 pints of fluid and the ureters were much dilated. The specimen was sent to the College by Dr. Rayner, who has briefly recorded it in the *British Medical Journal*, 1892 (ii, p. 1384).

¹ *Edin. Hosp. Reports*, 1896, iv, p. 116, and *Scot. Med. and Surg. Journ.*, June, 1897 (Bibliography); cited by Dr. G. F. Still, *Trans. Path. Soc.*, 1899, i, p. 96.

Dr. Rayner was, on November 27, 1892, sent for by the midwife of the Marylebone Workhouse, because she could not complete the delivery of a primipara. Upon examination of the child after it was delivered, the "urethra was perfect and allowed a probe to pass easily." "There was meconium in the first part or so of the jejunum only, none in the colon or rectum, both of which were so cord-like as to be easily taken for ureters, as were also the lower two-thirds of the small intestine." The external genitalia are normally developed, the lower limbs naturally disposed. A rod of glass 2 mm. in diameter at present lies in the urethra, and, as viewed from within the bladder, the internal meatus is quite normal; it is not patulous, as though the blockage lay further forwards, in the urethra. The second specimen is in the Museum of Guy's Hospital (No. 2,551/40). It consists of the lower part of a female foetus at term. The lower limbs are well developed and lie in a normal position. Labour was so impeded by the enlargement of the abdomen that the child was destroyed. The labia were adherent by a "thick mucus," but on their separation the urethra was found pervious. Some while ago I had the opportunity of examining this specimen; the hymen and other parts are normally developed, and on separating the labia the orifice of the urethra is brought into view; I could easily pass a probe into the bladder. There were no traces of any organic adhesion, and the presence of the thick mucus above referred to was possibly due to the absence of micturition and the non-clearance of the external parts. The case was observed in 1863 by Dr. Braxton Hicks and Mr. Leach.

From the foregoing evidence it is not improbable that the condition found in the adult described in the present communication was of infantile origin, as is the case in idiopathic dilatation of the colon. The result in both is chronic constipation, if one might extend the term to the retention of fluid and speak of a constipated bladder. It may be pointed out here that in Dr. Dawson Williams's case there was marked polyuria. The child, when admitted to the hospital, was passing large quantities of pale urine, which contained a trace of albumin. The dilatation of the ureters and the hydronephrosis were very pronounced, and in the foetal case in the College Museum the bladder is very greatly distended, as it may be when the urethra is imperforate, as though an excessive amount of urine had been excreted by the kidneys.

The occurrence of intra-uterine polyuria, it has been suggested, is really the factor that produces the dilatation of the bladder and the accompanying hydronephrosis. This method of viewing the sequence of events, however, cannot, I think, be substantiated. Polyuria in the

adult does not give rise *per se* to dilatation of the bladder and hydronephrosis when there is no obstruction to the passage of urine beyond. Far more probably the polyuria, such as was noticed in Dr. Dawson Williams's case, results from the hydronephrosis, and there being a persistent obstruction a vicious circle arises, in which the hydronephrosis brings about polyuria, and the resulting polyuria brings about a further increase in the hydronephrosis. For the passage of an abundant dilute urine is a recognized phenomenon in double hydronephrosis (J. Rose Bradford); and the trace of albumin noticed in the case just referred to may be attributed to the fibrosis of the kidney and the changes in the epithelium accompanying the compression of the renal substance.

The occurrence of hypertrophy of the bladder without organic obstruction is a phenomenon that was known to Paget, whose reference to it I may cite: ¹ "In some cases it appears certain that hypertrophy may occur without either phimosis, calculus, stricture, or any similar obstruction. It was so in a case illustrated in the Museum of St. Bartholomew's in a child aged 4, who had suffered intensely with signs of stone in the bladder, but in whom no stone existed; no disease of the urinary organs could be found, except this hypertrophy of the muscular coat of the bladder. An exactly similar case, in which after exceeding irritability of the bladder the enlargement of its muscular coat appeared the only change, was under Mr. Stanley's care. In such cases the too frequent and strong action of the bladder, though irritable and unhealthy, seems alone to give rise to hypertrophy of the fibres. It is, however, possible that the change may be due to temporary closure of the urethra by muscular action. If, for example, the compressors of the urethra, instead of relaxing when the muscular coat of the bladder and the abdominal muscles are contracting, are in the habit of contracting with them, the obstacle they would produce in the urethra will soon engender hypertrophy of the bladder. Certainly such cases of disagreement in the action of the bladder and urethra occur in adults, and they may be called cases of stammering bladders, for their phenomena, both muscular and nervous, are exactly parallel with those of ordinary stammering in speech."

Paget's short article upon "Stammering Bladder" was published in the *British Medical Journal*, October 24, 1868. In this he remarks that the patient can often pass his urine without any trouble, but at other times he suffers all the distress that he might have with a very bad

¹ "Surgical Pathology," 3rd ed., 1870, p. 57.

urethral stricture. "He cannot pass a drop of urine, or, after a few drops, there comes a painful check, . . . and complete retention may ensue and overfilling of the bladder. The stammering with the bladder occurs in just the same conditions as the stammering of speech. . . . The worst times of such patients are when with strangers, or with persons, or in places that are associated in their minds with stammering. The bladder, unable to expel its contents, becomes for a time the seat of the feelings of distress and tightness and urgent need of emptying which are felt in more simply mechanical retention of urine. In cases of long-standing urinary stammering, some of which begin in very early life and some of which I have known for many years, I have seen no indication of any supervening organic disease. After years of trouble nothing appears wrong but in the manner of action of the parts."

A different pathogenesis clearly obtains in different cases. Some would appear to come under the head of a hyperæsthetic condition of the mucosa, whereby an abnormal amount of muscular contraction was excited by what in health would be a normal stimulus, and one which would not act until the organ had reached its usual degree of distension; although it must be confessed that in the cases cited by Paget the influence of an irritating condition of the urine or of a bacterial infection cannot be excluded. Here we should have hypertrophy without dilatation.

In others there is dilatation with hypertrophy, and these appear to be due to want of coördination between the contraction of the detrusor and the dilatation of the sphincter, or, as Paget suggests, of the urethral muscles anterior to it. We are led to choose between this and a hypo-æsthetic condition of the mucosa, whereby the micturition-reflex fails to be excited until the viscus has reached what in normal circumstances would be an abnormal degree of fulness. The organ would in either case become habitually over-distended, but without any necessary atrophy of the muscular wall. Had there been a muscular obstruction in front of the prostatic portion of the urethra, the latter would probably have become dilated in the way seen in cases of long-standing stricture. But this has not happened in the specimen, and one is induced to believe, therefore, that the obstruction lay at the neck of the bladder.

That the possibility of a hypo-æsthetic state of the vesical mucosa from which the reflex starts is not to be ignored in explanation of idiopathic dilatation will appear from the results of certain experiments carried out by Dr. T. G. Brodie and myself, in which we rendered the mucous membrane of the bladder anæsthetic by the intravesical injection

of cocaine. Whether anæsthesia and hypo-æsthesia of the mucosæ occur as hysterical phenomena is a subject to which little attention has been directed, but it is one that would repay further investigation. That the pharynx may be so affected is almost the only observation extant in this connexion, but this is well recognized and "of great importance in the diagnosis of the disease (hysteria)." ¹

Dr. Parkes Weber has published such a case,² in which, associated with complete hysterical cutaneous anæsthesia, the back of the mouth and the pharynx were also anæsthetic. The patient was a well-built man aged 21, not apparently of excitable temperament. He could feel nowhere excepting over a small area of skin bordering on the genital organs; owing to the anæsthesia of the back of the mouth and pharynx, laryngological examination was peculiarly easy. Sensation gradually returned and he was discharged well. The anæsthesia disappeared last from the limbs; on these the anæsthetic areas were sharply limited as by a garter, a condition (as Charcot has pointed out) almost pathognomonic of hysterical anæsthesia.

In the cat Dr. T. G. Brodie and I have found that the result of cocainizing the interior of the bladder is to abolish micturition so long as the local effect of the drug lasts. After many other methods of experiment we arrived at that of using A. C. E. mixture as an anæsthetic in place of chloroform, or of pithing accompanied with destruction of the sensorium; and instead of opening the fundus of the bladder and filling the viscus from an elevated bottle of warm salt solution through a wide tube tied into it, we finally adopted the simpler expedient of passing a catheter by the urethra and limiting ourselves to the use of female cats. The bladder was filled with warm salt solution through the catheter, the latter being of such a size as to allow of micturition taking place around it. When the bladder is lightly filled for ten minutes with 5 per cent. solution of hydrochloride of cocaine, and the salt solution then introduced, micturition is abolished, the viscus becoming distended to a remarkable degree without dilatation of the sphincter and escape of fluid by the side of the catheter occurring. These results indicate that the micturition-reflex arises in the vesical mucosa, the tension of which excites both the detrusor to contract and the sphincter to dilate. This simple method was, in our later experiments, amplified by using a double catheter, the inner tube of which was connected with a manometer to

¹ Dr. S. J. Sharkey, *Brain*, 1904, xxvii, p. 1, Presidential Address before the Neurological Society.

² *St. Bart.'s Hosp. Reports*, 1898, xxxiv, p. 313.

measure the pressure of the fluid within the bladder, whilst the bladder itself was filled through a side channel in the outer tube. The details of these observations will be published later.

ADDENDUM.

Mr. C. E. Lakin has been good enough to furnish me with the short notes of a case of greatly distended bladder which was recently under the care of Dr. Essex Wynter in the Middlesex Hospital. The patient was a spare man, aged 64, who died with diabetes. On opening the abdomen after death an enormously distended bladder was found rising out of the pelvis; it occupied the whole of the right half of the peritoneal cavity and extended some distance beyond the middle line into the left side. It measured 15 in. in its greatest longitudinal diameter, and 12 in. transversely. Its summit was in contact with the lower surface of the right lobe of the liver, and the intestines were for the most part displaced to the left side of the abdomen. Rather more than 180 fl. oz. of urine were withdrawn from the bladder, the wall of which was of the usual thickness; there was no evidence of cystitis. The ureters and renal pelves were dilated. There was in this case a slight general enlargement of the prostate; and a prostatic collar, on which were two small projections, was visible from within the bladder. Clinically there was no incontinence and no difficulty of micturition; the patient had micturated like any other person. Mr. Lakin remarks that there was no evidence to show that the dilatation and hypertrophy were due to the prostatic condition discovered after death.

Pathological Section.

January 19, 1909.

Mr. S. G. SHATTOCK, President of the Section, in the Chair.

On some Hereditary Syphilitic Affections of the Nervous System.

By PIETRO RONDONI (Florence).¹

I AM indebted to the kindness of the Director of the Laboratory (Dr. Mott) for the opportunity of bringing before your notice, the histological study of three cases of hereditary syphilis with affection of the nervous system. Two of the cases were juvenile general paralysis, and the other syphilitic brain disease.

The brains were fixed in formol, and the sections were treated according to the usual methods (Nissl, Weigert, &c.). For glia I have found very useful and simple the method introduced by Dr. Ranke, of Heidelberg, published by him in the *Zeitschrift für die Erforschung und Behandlung des jugendlichen Schwachsinn*s, Bd. i., H. 2, 1906.

I will commence my remarks with a description of the two cases of juvenile paralysis. The first case showed typical characteristic changes in the teeth, and the clinical notes speak of a brother of the patient who is suffering from a similar form of the disease to that exhibited by this patient during life. A history obtained from Guy's Hospital concerning the father, who recently died there, enabled me to ascertain that he had suffered with syphilis thirty-four years ago, and that the conditions of his organs and vessels found post mortem accorded with this fact. The patient was aged 18 at the time of death; had always been considered as an imbecile, and only two years before death occurred the symptoms of the present illness came on and were very characteristic—viz., presence

¹ From the Pathological Laboratory of the London County Asylum, Claybury.

of Argyll-Robertson's sign, inequality of pupils, tremors, alteration of the speech, inco-ordination of arms, unsteady gait, great psycho-motor activity, a progressive loss of never much developed intellectual faculties. We have thus a symptomatology which occurs sometimes in cases of the adult form of the disease—viz., a progressive dementia and paresis without delusions. It is often found, if we look at the literature of this subject, that juvenile general paralysis chooses frequently children who already

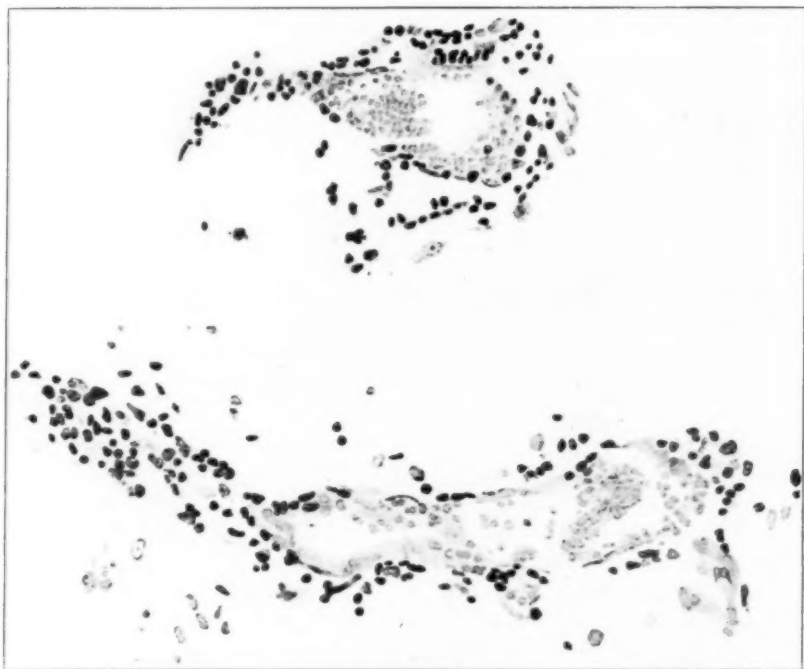


FIG. 1.

Infiltration around the vessels in the basal ganglia (Case I). Thionin. Magnification 350.

present some characters of deficiency in the development of the intellectual faculties, and sometimes children who show a very deep degree of idiocy: it often seems that a condition of diminished resistance of the brain acts as a predisposing cause, and this condition of greater debility of the nervous system may be due to an arrest of development which

may find an anatomical expression in microscopic changes in the brain. I have recently had the opportunity of studying in the laboratory of Professor Edinger, in Frankfort-on-the-Main, the brain of a young child which exhibited arrest of development of the cortex associated with the characteristic changes of general paralysis. One of the chief histological characters of general paralysis is the perivascular infiltration with plasma-cells and lymphocytes especially marked in the cortex; the interesting fact in this case is that I discovered these changes most marked in the basal ganglia and white matter between them. I would therefore suggest the desirability of a systematic examination of the basal ganglia in all cases of general paralysis, for it may happen that these structures may show changes of a similar nature to this in a larger



FIG. 2.

Vacuolization of the cells of Purkinje in juvenile general paralysis (Case I). Thionin.
Magnification 600.

number of cases than is now anticipated. Proliferation of vessels and rod-cells were also present in the cortex. The degenerative changes in the cortex were fairly marked, and they are certainly responsible for the greatest part of the symptomatology.

In the second case we have a very interesting family history which shows the progressive attenuation of the syphilitic virus on the offspring: Father syphilitic; six children; the first two, born before term, died within a few hours of birth; the third shows keratitis and otitis with deafness; the fourth is the patient; the last two are well and grown up to bright children. The clinical symptomatology consists of a progressive impairment of the conditions of mind and body, with many epileptic fits,

and a tendency to spastic phenomena as it is often met with in the juvenile form of paralysis; the pupils were unequal and reacted sluggishly to light. The diagnosis admits of no doubt, because the macroscopic changes found post mortem, as well as the changes which the microscope reveals, are most characteristic, and are much better marked than in the first case. The microscopic changes show numerous large plasma-cells in the adventitial sheaths of the vessels of the cortex, the pia being also infiltrated; some rod-cells are present; proliferation of the small vessels with increased vascularization of the brain and degenerative changes in the walls of the vessels were also marked. The degenerative changes in the nervous elements of the brain were scattered everywhere.

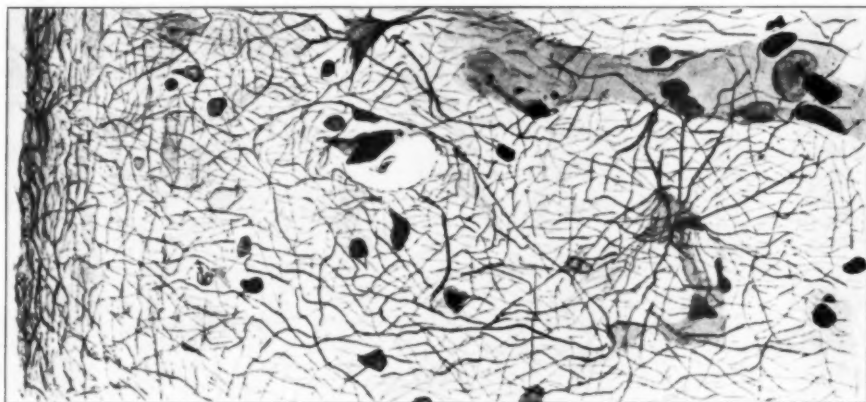


FIG. 3.

Proliferation of glia at the surface of the cortex; spider-cells, footlike processes, &c.
(Case II). Ranke. Magnification 600.

I would also call attention to the very typical changes of the glia both in the cerebrum and in the cerebellum—viz., large spider-cells, thick felt-work at the surface of the cortex, tendency of the proliferating glia to increase the limiting surfaces with formation of little footlike prolongations towards the vessels (Held) in the cerebrum; increase of the vertical Bergmann's fibres, formation of a marginal border (not present normally), presence of large astrocytes in the place of many of the cells of Purkinje in the cerebellum. I have found in the oblongata and spinal-cord well-marked degeneration of the pyramidal tracts, which accounts for the spastic phenomena.

I may remark that in both these cases I have found a particular vacuolization of the cells of Purkinje in the cerebellum; such degenerative change seems, according to Straüssler (and I agree with him),¹ to have some diagnostic importance without being of course specific.

The third case is of a different nature to the foregoing cases. The family history is very similar to that of the second one: there occurred two miscarriages; one boy living seven months, one boy living fifteen months; then the patient, who died when aged 23; then came a healthy living girl; then a girl who died when aged 16 months after the death of the mother. The father does not admit having had syphilis. Both parents were alcoholic. However, it is much more likely that the numerous miscarriages and death of children were due to a not admitted syphilis than to alcoholism, otherwise we cannot understand the progres-

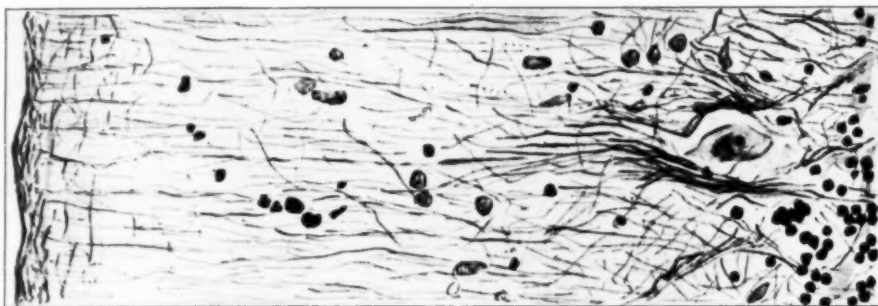


FIG. 4.

Proliferation of glia in the cerebellum: increase of vertical fibres, formation of a marginal border, &c. (Case II). Ranke. Magnification about 200.

sive improvement in the result of conception, for if the alcoholic habits were the sole cause then the devitalizing influence should have progressed instead of improved; in fact, this history agrees so strongly with that of other known syphilitic cases that it may be assumed that it was of syphilitic origin, especially when the facts of the investigation of the cerebral vessels are correlated with the above history.

The patient, a girl, was healthy until aged 14; from thence progressive dullness and apathy, later fits (apopleptic), coarse tremor of arms, some nystagmus, exaggeration of knee-jerks, and inequality of pupils. Histological examination revealed two forms of morbid changes: one diffuse degeneration of the cortical neurons without any specificity and in

a not very marked degree; the other changes in the vessels of the base of the brain, which can be considered as a diffuse arteritis with predominance of the lesions in the intima. These vessels—for instance, the small arteries which arise from the Sylvian and go to the basal ganglia—have thickened walls, sometimes with formation of small aneurysms; and in these walls we see increased connective, also elastic, tissue, with degenerative appearances, the demarcation of the various coats being



FIG. 5.

Calcareous infiltration of the vessels and nervous tissue in the nucleus lenticularis on right side (Case III). Magnification 40.

sometimes obliterated by the irregular alternation of degenerative and proliferative changes. I found in the basal ganglia some calcareous infiltration of the small vessels, the media seeming to be preferred, and some old patches of softening, which probably can be correlated with the apoplectic fits. The small veins are also attacked. Is this vascularitis a syphilitic one? If we take into consideration the following facts—

the absence of patches of atheroma, the marked affection of the elastic tissue, some slight infiltrations with lymphocytes and plasma-cells around the adventitia of some of the arteries—we may conclude, without concealing the difficulty of distinguishing an *endoarteritis syphilitica* from other forms of *endoarteritis obliterans* or *deformans*, that we have in this case, I think, to do with a form of *syphilis hereditaria tarda* of the nervous system similar to the cases of Homén and of De la Chapelle.

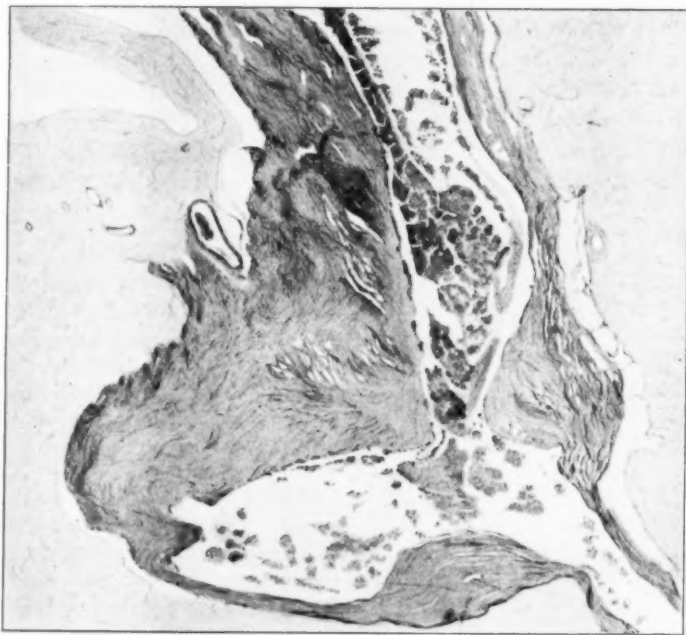


FIG. 6.

Changes in an artery in the basal ganglia, showing the formation of a small aneurysm (Case III). Magnification 50.

The cases of Homén show also diffuse degenerative changes in the cortical cells, without granulations in the ependyma, with arterial lesions and softening in the basal ganglia, with only very slight infiltration of the adventitial sheaths and with very little proliferation of glia. The cases of Homén differ only because they were familial (five brothers and sisters). Supporting the diagnosis of *syphilis hereditaria tarda* are also

the anatomical conditions of the ovaries, which suggest an arrest of development in early stages and give to the case the stamp of infantilism, one of the *stigmates distroffiques* of the *syphilis hereditaria tarda* according to Fournier; such characters of infantilism were found by Mott in a number of his cases of juvenile general paralysis. At any rate, progressive general paralysis must be excluded in this case by reason of the fact that the histological changes which nowadays are considered as necessary for the diagnosis of general paralysis are absent—viz., perivascular infiltration with plasma-cells and lymphocytes (with predominance of the former), the existence of rod-cells, marked neuronie decay and characteristic glia proliferation, as we know from the classic researches of Nissl and Alzheimer in Germany, and of Mott and Watson in England.

The extensive bibliography on the subject of "Hereditary Syphilis of the Nervous System and Chiefly on Juvenile General Paralysis" I shall refer to in a work which will soon appear in the *Archiv für Psychiatrie*.

Dr. F. W. MOTT, in answer to the President, said that when he was working at sleeping sickness he concluded that there were appearances which suggested that the plasma-cells might come from the endothelial cells, or else from the lymphocytes, because he was able to trace the transition from lymphocyte to plasma-cell through all the stages, and he had seen the same appearances in general paralysis of the insane. He considered that the existence of plasma-cells around the vessel was very suggestive of syphilis or trypanosome affection, and he had not met with plasma-cells to any extent in any diseases except sleeping sickness, syphilis and general paralysis. Vogt said plasma-cell infiltration was diagnostic of general paralysis, but he had not seen sleeping sickness. In order to show the value of that observation he mentioned that he had fifty blocks, cut from various cases of brain disease, in the laboratory, and he asked his assistant, who had the register of the specimens, to stain them and put them under microscopes, and he (Dr. Mott) would pick out the cases of general paralysis and sleeping sickness. From the plasma-cell and lymphocyte perivascular infiltrations he succeeded in picking out correctly the thirteen cases of general paralysis and two cases of sleeping sickness. He regarded the interest of the present paper as twofold because of the discovery of the perivascular infiltrations of the basal ganglia in one of the two cases of general paralysis. He rather expected those changes because in sleeping sickness one found the infiltration in that region was even more marked than in the cortex, and that was not usually so in general paralysis. General paralysis was a wasting disease of the whole nervous system, but chiefly of the cortex. The second important point in the paper which was of great practical value was the simple method of staining the neuroglia described by the author. Those who had used the Weigert method, which is brilliant when it comes off, but does not always succeed, would appreciate the present one. He felt very grateful to Dr. Rondoni for bringing forward the method.

Disseminated Lobular Necrosis of the Liver with Jaundice
(*Hepar Necroticum cum Ictero* of Curschmann and H. Oertel), and a Case of Acute Hepatic Atrophy in Secondary Syphilis.

By F. PARKES WEBER.

(I) DISSEMINATED LOBULAR NECROSIS OF THE LIVER.

D. S., AGED 35, a shoemaker, was admitted at the German Hospital on February 25, 1908. At first he was on the surgical side under Dr. Michels, but was afterwards transferred to the medical side under Dr. Parkes Weber. The present illness was said to have commenced gradually sixteen months before admission with pains, vomiting and jaundice. The jaundice had persisted since then, though the pains and vomiting had left him. For the last four weeks he thought that he had had fever. There was no history of syphilis or other previous diseases.

On admission the patient was deeply jaundiced, of a greenish colour, not very emaciated. His faeces were colourless. His urine was bilious, of specific gravity 1022, acid, free from albumin, but yielding a cloud of nucleo-proteid with acetic acid. There was ascites, and the liver could be felt below the right costal margin. The spleen could not be palpated. There was considerable fever, the temperature rising to 102° F. in the evenings. On February 28 a small incision was made and the ascitic fluid evacuated; nothing abnormal was felt, except that the liver was enlarged. After this there was less fever in the evenings, and after March 5 there was no fever at all. But the patient continued to lose weight and strength, and became greatly emaciated. The ascitic fluid accumulated again, and was evacuated by paracentesis on March 7. *Blood examination* (March 9, 1908): Hæmoglobin (by Haldane's method), 55 per cent.; red cells, 2,000,000 in the cubic millimetre of blood; white cells, 11,920. Microscopic examination of a blood-film showed polymorphonuclear leucocytosis; one nucleated red cell was seen. The urine (March 9) was bilious, of specific gravity 1019, and free from albumin and sugar. Death occurred on March 12.

The *post-mortem examination* was practically confined to the abdominal organs. A hard tumour of the size of a walnut was found in the

head of the pancreas pressing on the common bile-duct. The gall-bladder and all the extra-hepatic and intra-hepatic bile-ducts were dilated with green bile. The liver was enlarged, weighing 91 oz., and was greenish in colour. Pieces were removed for microscopic examination. There were several moderately enlarged lymphatic glands in the neighbourhood of the head of the pancreas and in the mesentery, but otherwise no metastatic tumours were seen. The spleen was somewhat enlarged, weighing 16 oz. The kidneys did not appear diseased.

*Microscopic Examination.*¹—The growth in the head of the pancreas was carcinomatous, and the enlarged lymphatic glands were found to be secondarily infiltrated. The splenic swelling seemed to be due, at all events in part, to engorgement of the organ with blood.

The Liver.—Microscopic sections showed a decided increase of interacinous tissue, fibrous rather than cellular, constituting a kind of sclerosis of interacinous (monolobular or rather unilobular) distribution. Within the acini the columns of liver-cells appeared somewhat shrivelled and abnormally separated from each other, the spaces between them being occupied by dilated blood-capillaries. Many of the liver-cells and bile capillaries were crammed with bile pigment (or rather, inspissated bile). But what chiefly attracted one's attention was the presence in the hepatic parenchyma of multiple, scattered, sharply defined, circular "islands" of tissue which had undergone some necrotic change (*see plate*). These necrotic islands mostly occupied the whole of a lobule (acinus); their average diameter probably equalled that of the lobules. The liver-cells in these areas seemed to have faded or "melted away," leaving a scanty "cobwebby" meshwork of degenerated ground substance (capillary walls, &c.), containing a few nuclei and fat globules. In the central portions of these areas the necrotic debris was often either deeply stained with bile pigment, apparently by imbibition, or evidently replaced by an actual extravasation of bile from the over-distended bile capillaries. In some of the necrotic areas, however, especially in their central zones, a subsequent small cell-infiltration had succeeded the changes just mentioned. The central portions of these areas were filled with round cells, and the bile-stained debris had been more or less completely absorbed or formed a narrow zone around the central mass of round cells. Outside this zone there was a clearer zone occupied by the meshwork left by the atrophied liver-cells—*i.e.*, consisting of degenerated remnants of capillaries, fat globules, &c. Outside this

¹ For the microscopic sections in this case I am greatly indebted to Mr. E. H. Shaw, and also to Dr. Chapuis, one of the House Physicians at the German Hospital.



FIG. 1.

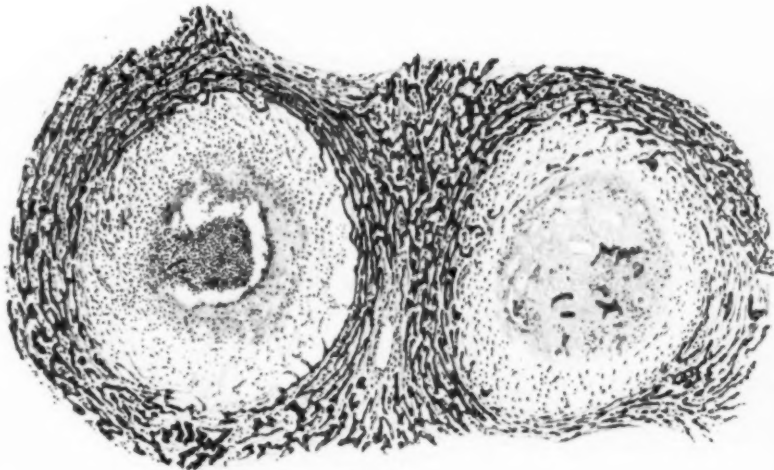
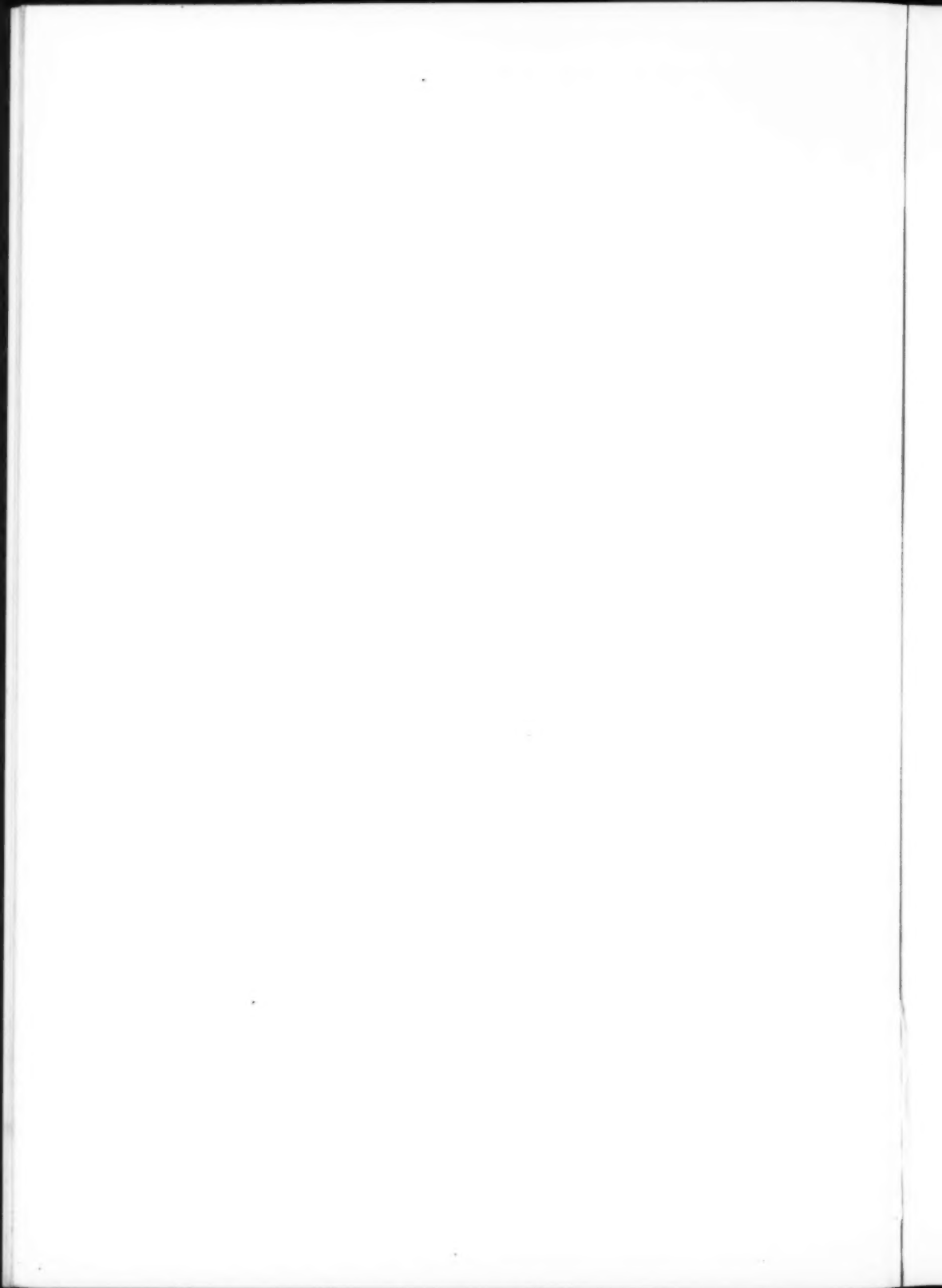


FIG. 2.

The figures (magnification $\times 60$) show several of the necrotic areas in the liver. In two of them (one in each figure) the central bile-stained debris has been partially replaced by small cell infiltration. The stains employed were Jenner's eosinate of methylene blue for figure 1, and Loeffler's alkaline methylene blue for figure 2.



again, and enclosing the whole island of necrosis, there was usually a more or less complete capsule formed from the surrounding tissue, especially by the interlobular fibrous tissue already referred to. In specially stained sections no microbes could be discovered, either in the necrotic areas or elsewhere. The small cells of the areas of cell-infiltration mostly contained irregularly shaped, branched, or broken up nuclei.

I cannot here enter into the whole subject of focal, zonal and diffuse necroses of the liver, a subject which has been much investigated, especially in America (Walter Reed, F. B. Mallory, S. Flexner, E. L. Opie, R. M. Pearce, Horst Oertel, J. McCrae, and O. Klotz, &c.), both by post-mortem observations in human beings and by experimental researches in animals. The small focal necroses, which as seen in the later stages of cellular infiltration have been termed "lymphoid nodules," chiefly met with in typhoid fever cases, differ considerably from the relatively large scattered islands of necrosis found in our case, but hepatic lesions in jaundiced patients identical with those in our case have been carefully described by H. Curschmann (1899)¹, and Horst Oertel (1904 and 1906)². Curschmann described the process as a "peculiar form of necrosing hepatitis"; whilst Oertel, though giving all due credit to Curschmann, described it as a "multiple non-inflammatory necrosis of the liver with jaundice (*hepar necroticum cum ictero*)."³ I would prefer to term it a "disseminated lobular necrosis of the liver," since there is a decided tendency for the necrotic process to affect whole lobules, in which respect it differs from the ordinary focal necroses of typhoid fever.

In Curschmann's first case the patient was a woman, aged 51, with vomiting, jaundice, irregular fever and bed-sores. At the necropsy the liver was seen to be of dark brown colour and about usual size. A rough ("mulberry") calculus obstructed the common bile-duct, which was dilated behind the obstruction. Between the gall-bladder (which was contracted) and the liver was an encapsuled abscess filled with bilious purulent matter and detritus. The whole liver substance was studded with minute (miliary to pea-sized) spots. Microscopic examination of different parts of the organ showed a disseminated necrotic change tending to be of centro-acinous distribution. Bile-imbibition in

¹ Curschmann, "Ueber eine eigennartige Form von nekrotisirender Hepatitis," *Deut. Arch. für Klin. Med.*, Leipzig, 1899, lxiv., p. 564.

² H. Oertel, *Journ. Med. Research*, Boston, 1904, xii, p. 75; and *Journ. Exper. Med.*, New York, 1906, viii, p. 103.

the necrotic areas was a striking and characteristic feature. The minute (miliary) spots visible to the naked eye were due to a whole acinus being filled with bile-stained necrotic material. The largest spots (those up to the size of a pea) were apparently due to the fusion of several necrotic acini. There was no true suppuration in the hepatic parenchyma. The portal spaces in Curschmann's case showed a decided sclerotic change, as they have done in most of the cases subsequently examined.

The description of Curschmann's two other cases and Oertel's four cases help to confirm and elaborate the features of this peculiar liver change. More or less jaundice and a toxic condition of some kind or other are probably essential pathogenic factors in the production of the characteristic microscopic appearances. In our case a toxæmic factor was evidenced during life by the intermittent pyrexia. Probably the degeneration of the hepatic cells in these cases, a form of degeneration which Oertel would like to term "cytolysis," commences usually but not always in the central region (centro-acinous zone) of the affected acinus, but soon affects the whole acinus, and several neighbouring acini may become necrosed and coalesce, as described in Curschmann's first case. The central portion of each necrotic "island" is generally at first deeply stained with bile pigment, by imbibition or extravasation of bile, but afterwards is infiltrated with round cells which clear away or replace debris and bile pigment. Sections through a necrotic "island" at this stage show a central area of small cell-infiltration, around which is a narrow zone of bile-stained debris. Outside this in our sections is a pale zone, which appears to be occupied by a mesh of "ground-work" substance (degenerating capillary walls, &c.), from which the hepatic cells have faded. A kind of capsule, made up of compressed hepatic parenchyma and interacinous fibrous tissue, constitutes the outermost zone of the necrotic islands. The margins of these necrotic areas are always sharply defined. More or less interacinous sclerosis seems to be generally present, and in some cases there may have been a certain amount of preceding cirrhotic change. Our case differs somewhat from the others in that the jaundice was due to cancerous obstruction of the common bile-duct, but it seems to us that the essential factors in the production of the histological picture in all these cases are: (1) some toxæmic condition giving rise to patchy necrosis of the hepatic parenchyma; (2) increase of bile-pressure (from some kind of obstruction to the flow of bile) favouring extravasation of bile into the necrosed areas.

(II) ACUTE HEPATIC ATROPHY IN EARLY OR SECONDARY SYPHILIS.

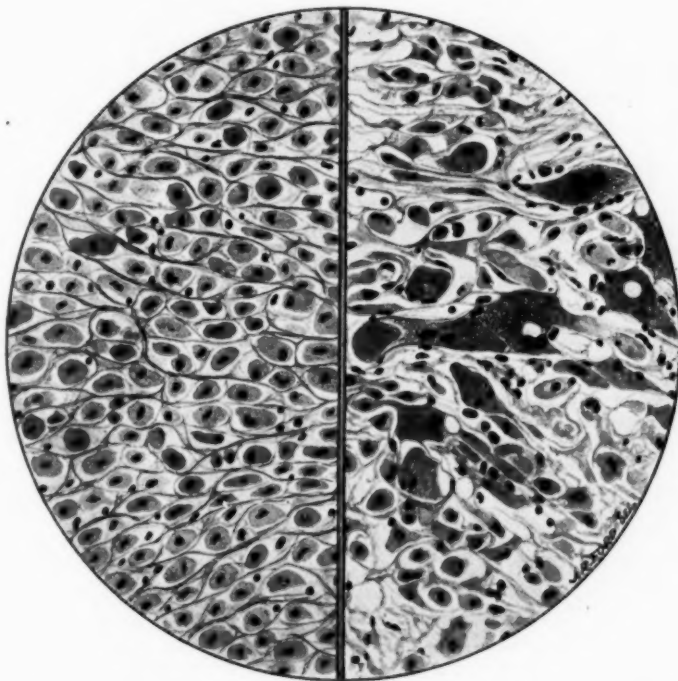
M. V., aged 22, was admitted to the German Hospital in the afternoon on March 25, 1902, under the care of Dr. Fürth. He was a tolerably well-nourished man of medium size, a German waiter in London. The history was that two months previously he had had a hard chancre, for which he had been treated by mercurial injections. The jaundice from which he was suffering had commenced fourteen days before admission. During the last days he had felt worse, but was able to walk to the hospital. Soon after admission, however, he became mentally dull and drowsy. His temperature was 97° F. and his pulse about 68 in the minute. During the following night he vomited, uttered peculiar cries and became unconscious. On the next day (March 26) he was passing urine and fæces into the bed. The limbs were rigid and the knee-jerks were excessive. Temperature 96° F. The jaundice was very deep. There was no pain by palpation in the hepatic region. The liver could not be felt. A hundred cubic centimetres of blood were removed by venesection from the right median cephalic vein and two litres of normal salt solution were injected subcutaneously. On the following night there was Cheyne-Stokes respiration and recurrent tonic contraction of the muscles of the limbs with trismus. The patient died early in the morning of March 27. The urine passed on March 25 was very bilious, slightly acid, of specific gravity 1015, free from sugar, but apparently containing a trace of albumin or nucleo-proteid; in the sediment some bile-stained tube-casts were seen, but no crystals of leucin or tyrosin.

Necropsy.—The brain, except for jaundice, presented no macroscopic evidence of disease. The heart weighed 10 oz. and appeared normal except for some ecchymoses below the epicardium; the cardiac muscle was pale. Nothing abnormal was noted in the lungs, kidneys, pancreas, or alimentary canal. The spleen weighed about 9 oz. and was rather soft ("pulpy"). The liver was decidedly too small, weighing only 39 oz., and was too flaccid. The capsule could be "crinkled up," as in cases of atrophy of the hepatic parenchyma, but it appeared otherwise normal. The biliary passages were free. The cut surface of the organ had lost its characteristic normal markings, but the division into red and yellow areas, so often seen in acute atrophy, was not noted. Microscopic sections from two portions of the organ showed no normal hepatic parenchyma. The lobular arrangement could, however, be still made out by using the

blood-vessels as landmarks. In some parts, especially in the marginal zones of the lobules, the liver-cells had practically disappeared, leaving only a meshwork of "ground substance" ("Stützgewebe," made up of the walls of the blood-capillaries, &c.). In most parts the hepatic cells seemed shrivelled, with their nuclei small and pycnotic or altogether absent—that is to say, apparently absent—because they had undergone karyolysis, or at all events were not differentiated from the cytoplasm by the stains employed; the cells appeared to be lying in but not filling compartments, separated from each other by the meshwork of the "ground substance," or "supporting tissue," already mentioned. Some of the cells contained globules, probably fatty in nature, but no special stains for fat were employed. In other parts a more acute process of degeneration of the parenchyma was indicated, the cells were necrosing, often apparently without preliminary shrinkage; their cytoplasm had undergone a fine granular ("woolly") change, and their nuclei were fading (that is to say, stained badly), or had already disappeared (karyolysis), or were breaking up and setting free the chromatin-particles (karyorrhexis) (*see figure*). The bile canaliculi generally appeared to be increased. In some parts there was very little evidence of inflammatory reaction, but in other parts there was considerable small cell-infiltration around the interlobular vessels; but there was no evidence whatever of preceding cirrhosis or any kind of earlier disease in the liver. At some spots, chiefly centro-acinous in distribution (*i.e.*, around the central veins of the lobules), were groups of fairly well-preserved liver-cells, and some of these contained more than one nuclei, as if an attempt at cell-regeneration was already in progress. No vascular changes were observed, such as are associated with the lesions in various organs met with during later periods of syphilis. An old section of the liver was kindly re-stained (Levaditi method) for spirochaetes by Dr. J. C. G. Ledingham, but with negative results.

A benign form of jaundice is well known to occasionally associate itself with the ordinary symptoms of secondary syphilis, accompanied perhaps by a little fever, but soon passing off with (or possibly without) antisypilitic treatment. In two of the cases referred to by Arnheim jaundice accompanied the secondary rash and recurred during a relapse with condylomatous sore throat. A malignant type of jaundice—that is to say, "icterus gravis"—though much rarer than the benign form, has likewise been repeatedly observed during secondary syphilis. At first the jaundice may not appear very threatening, but then come great

weakness, vomiting, cerebral symptoms (stupor, delirium, convulsions, coma), and rapid diminution in the size of the liver, and death soon follows. In all such cases in which a post-mortem examination has been made a condition of hepatic atrophy has been found present, usually described as acute yellow (or red) atrophy of the liver. The number of such cases already described is so considerable that in my opinion it is



Showing, in the observer's left half of the figure, shrinkage of the hepatic cells, which lie in a fine mesh of ground substance. In the right half of the figure some of the cells retain their size, but are in various stages of granular change. (Magnification, Zeiss, obj. D, oc. 4.)

impossible to doubt that a causal connexion of some kind exists between the atrophy of the liver and the syphilis, even though the hepatic disease does not seem to be associated with the local presence of spirochaetes in the affected organ. In some cases of icterus gravis in secondary syphilis the liver has shown relatively chronic changes in addition to the

acute degeneration of the parenchyma. Thus, in Hilton Fagge's case (a woman aged 23) and in Siredey and Lemaire's (a girl aged 19) there was much cell-infiltration and "fibroid" change, said to recall appearances met with in the pericellular cirrhosis of congenital syphilis.¹ Similar hepatic changes were met with by H. D. Rolleston in A. H. Wilson's case, a girl aged 17, with a secondary syphilitic roseola, who became jaundiced six weeks before her death; her mental condition was affected during the last three weeks, and leucin and tyrosin were present in the urine at the end. Icterus gravis with acute parenchymatous degenerative changes in the liver may doubtless also occur during later stages of syphilis, as it did in a boy with congenital syphilis, aged 9½ years, whose case was recently described by F. J. Poynton; but with such cases we are not at present concerned.

According to W. J. Calvert, the occasional association of jaundice with syphilis was already noted by Paracelsus in 1510, and according to Neumann, Ribeiro Sanchez (1699-1783) believed that the jaundice was causally connected with the syphilis in such cases. That such a causal connexion of some kind exists in regard to the jaundice of secondary syphilis ("icterus syphiliticus") has in modern times been long maintained in France—namely, by Ricord, Gubler, Lancereaux, Cornil, Mauriac, Fournier, &c., though the actual proportion of cases showing the association is not great. S. Werner (1897), in 15,799 cases of early syphilis, found that jaundice was observed in only fifty-seven (that is to say, in only 0.37 per cent.), and according to O. Goldstein (1904) jaundice was noted in only twenty out of 7,462 early syphilitic cases (that is to say, in only 0.26 per cent.) in E. Finger's clinic at Vienna. It is quite possible, however, that slight jaundice in secondary syphilis is rather commoner than these statistics would seem to show.

Cases of "icterus gravis" and acute atrophy of the liver in secondary syphilis are, of course, much rarer, but, as I have already mentioned, many examples have been recorded. It was a paper by Engel-Reimers in 1889, with a careful report on three cases, which first seems to have attracted attention to the subject on the Continent. Senator, at the German Medical Congress of 1893, added accounts of two more cases, and in the discussion on Senator's paper Naunyn spoke of another case. In 1895 Meder succeeded in collecting accounts of twenty cases of acute

¹ Diffuse or circumscribed areas of pericellular cirrhosis recalling the changes met with in inherited syphilitic cirrhosis of infants have been occasionally noted in livers from cases of acquired syphilis in adult life. *Vide* Adami, "On the Stages and Forms of Syphilis," *Montreal Med. Journ.*, June, 1898; and F. P. Weber, "Diffuse Syphilitic Change in the Liver," *Trans. Path. Soc. Lond.*, 1899, 1, p. 42.

atrophy of the liver in secondary syphilis, so that Quincke, in his article on Diseases of the Liver in Nothnagel's great "System," vol. xviii, wrote: "In the secondary stage of syphilis, particularly at the commencement, acute atrophy of the liver has been sometimes recorded, following the jaundice which may occur at that period."

In 1898 P. F. Richter was able to collect forty-one cases, to the admission of some of which Veszpremi and Kanitz have recently objected, wrongly, I believe, in regard to the case described by J. Andrews (a man aged 20, who contracted syphilis five months before his death from acute atrophy of the liver) and Hilton Fagge's case (a woman aged 23) in the *Transactions of the Pathological Society of London*, 1866-67, xviii, p. 136. I find, however, that Richter did actually make two cases out of Hilton Fagge's single case, apparently having seen an abstract in which the patient's age was given as 32, instead of the real age, namely, 23 years.

Amongst other observations are those by Talamon (1897, a girl aged 17), Goldscheider and Moxter (1898, a girl aged 18), Thurnwald (1901, a man aged 24), van Niel Schuuren (1905, a woman aged 29), Nikolski (1906, a girl aged 16), and C. Fletcher (1906, a man aged 21). In regard to the last case Fletcher himself does not attribute the hepatic disease to the syphilis, but he mentions it as a fact that the patient had acquired syphilis six months before his death from acute yellow atrophy of the liver. Miller and Hayes, likewise, in their recently published investigation, mention that the patient (a girl aged 19) was supposed to have had a secondary syphilitic rash about five months before she died of acute hepatic atrophy.

In 1905 came the famous discovery by F. Schaudinn and E. Hoffmann of the (*Spirochæta pallida*) (*Treponema pallidum*) as the probably essential cause of syphilis, and as this parasite was found to be present in children's livers¹ affected by congenital syphilis (congenital syphilitic cirrhosis), it was supposed that it might likewise be present in the livers from adults with icterus gravis and acute hepatic atrophy in secondary syphilis. In all such cases, however, the search for the local presence of spirochætes in the liver has given a negative result. Buraczynski (1907) searched in two cases, Veszpremi and Kanitz (1907) in one case; also W. Fischer (1908, a man aged 22), P. Bendig (1908, a

¹ That the liver is one of the chief sites of the *Spirochæta pallida* in cases of inherited syphilis was demonstrated soon after the discovery of the organism in question—see especially, amongst the more recent papers on the subject, J. McIntosh, "The Occurrence and Distribution of the *Spirochæta pallida* in Congenital Syphilis," *Journ. of Path. and Bact.*, Cambridge, 1909, xiii, pp. 299-247.

girl aged 17), and A. Sézary (1908, a woman aged 25), each in one case; but always with negative results. Moreover, three apes of the genus *Macacus* were inoculated with pieces of the liver, spleen and bone-marrow from Fischer's case, but likewise with negative result, though later on a positive result was obtained in these same apes by inoculation with syphilitic condylomata lata. Altogether, up to the present time, fatal acute hepatic atrophy has been recorded in about fifty-three or fifty-four cases of secondary syphilis.

This brings us to the question of the *pathogeny of jaundice and acute hepatic atrophy in cases of secondary syphilis*. That there is some connexion between the liver affection and the syphilis one can hardly doubt, and it is quite possible that in many of the cases published as examples of idiopathic acute yellow atrophy of the liver a preceding syphilitic infection has escaped detection. Many still think that acute atrophy of the liver cannot be connected with syphilis, and they are therefore less likely to search for evidence of the latter. It is probable that the icterus gravis, accompanied by acute hepatic atrophy in these cases, is to be regarded as an exaggeration of benign "icterus syphiliticus"; and that the jaundice in such cases is really causally connected with the syphilis is evidenced by the cases (already referred to) in which persons whose earliest syphilitic exanthem was accompanied by jaundice became again jaundiced when they suffered from a secondary syphilitic relapse.

Amongst the theories that have been, or may be, suggested to explain the occurrence of mild or grave jaundice and acute hepatic atrophy in secondary syphilis are the following: (1) That the jaundice is of nervous origin ("emotional jaundice") and due to the mental worry and anxiety connected with the syphilitic infection; (2) that the jaundice is due to syphilitic enlargement of lymphatic glands, pressing on the extra-hepatic bile-ducts; (3) that the jaundice is due to venous congestion; (4) that the jaundice is due to a specific duodenal catarrh obstructing the bile-flow; (5) that the jaundice is due to a kind of syphilitic exanthem or erythematous swelling of the lining of the bile-ducts, analogous to the cutaneous roseola of secondary syphilis; (6) that the jaundice is due to obstruction of some kind in the biliary canaliculi; (7) that the hepatic atrophy is due to a specific change in the small blood-vessels; (8) that the hepatic atrophy is brought about in some way by the local presence and local action of spirochaetes in the liver; (9) that the hepatic atrophy is due to toxins produced elsewhere in the body by the vital or metabolic activity of spirochaetes and carried to the liver

in the blood-stream; (10) that in some cases it is due to the action of the mercurial treatment in addition to that of the (hypothetical) syphilitic toxin; (11) that it is due to counter-infection of the liver with *B. coli* or other microbes at a time when the resistance of the hepatic parenchyma is temporarily depressed by the syphilitic toxin circulating in the blood.

Simionescu refers to observations of Lioubimow and others showing that an enlarged lymphatic gland in secondary syphilis may actually press on the common bile-duct and give rise to obstructive jaundice. Naturally very few post-mortem examinations have been made in cases of secondary syphilis with jaundice (other than "icterus gravis"), and doubtless in many cases the jaundice is partly obstructive in origin and the fæces may be more or less acholic, but at least in some cases, according to O. Goldstein and F. Samberger, the fæces do not lose their colour, and bile still enters the duodenum.

In regard to the onset of "icterus gravis" with acute atrophy of the liver during secondary syphilis the theories which seem to suit best are those which I have numbered 7, 8 and 9. Though Tileston has recorded a case of acute hepatic atrophy in an apparently non-syphilitic boy after mercurial treatment, I do not think there is much to be said in favour of his suggestion that mercurial treatment (with or without the help of syphilitic toxin) plays a part in the causation of acute atrophy of the liver in syphilitic cases. If this were so, one ought almost to regard the appearance of jaundice in early syphilis as a contra-indication for mercurial treatment, whereas W. J. Calvert concludes his paper on "Icterus in Secondary Syphilis" (1904) as follows:—"Even the grave cases if properly treated are rarely fatal. Treatment consists in ordinary treatment for syphilis in this stage."

In regard to theory seven I believe that no evidence of specific disease of the hepatic blood-vessels has been discovered in the fatal cases, certainly not in the recently examined cases of Buraczynski, Veszpremi and Kanitz, and Sézary.

In regard to theory eight it has been already stated that in several cases diligent search for the local presence of spirochætes in the liver gave a completely negative result. In the present case likewise spirochætes were apparently absent from the liver, if the results obtained by re-staining old sections can be relied on.

Theory nine, which is upheld by recent authors, seems on the whole to afford the most probable explanation. It is maintained, in fact, that in secondary syphilis a toxin is produced by the spirochætes in the skin,

&c., which is carried to the various parts of the body by the blood-stream and that this toxin may more or less readily (in particular individuals) give rise to parenchymatous degenerative changes in viscera, such as the liver¹ or kidneys.

It would furthermore appear that the livers of women are far more susceptible to the harmful action of this hypothetical toxin than are those of men, since of those who succumb to acute atrophy of the liver in secondary syphilis there are about four females to one male. A possible analogy may be found in rheumatic chorea if that disease be regarded as a manifestation of the injurious effect of a toxin on the brain—that is to say, of a toxin produced by the rheumatic microbes in the joints or elsewhere and carried in the blood-stream to the brain. One would then have to suppose that the brains of girls were more sensitive (vulnerable) to that particular toxin than the brains of boys.

In regard to the term "acute" atrophy of the liver it appears from microscopic examination that the rapidity of the process varies in different cases and in different parts of the same liver, and that the process is not always so rapid as the acuteness of the final clinical symptoms ("icterus gravis") would lead one to suppose.² In some parts of an affected liver the parenchymatous cells seem to have had time to shrink or show fatty degeneration, whilst in other parts they have undergone a more rapid change, an acute necrosis of the parenchyma having occurred. In some cases (Siredey and Lemaire, &c.) a more chronic or fibroid change (preceding the icterus gravis doubtless) seems to have been present, a kind of intercellular sclerosis somewhat resembling the changes in congenital syphilitic cirrhosis, and one case (a woman aged 20) is recorded by Neumann in which apparently an acute (or subacute) atrophy of the liver in secondary syphilis was followed by regenerative changes in the form of adenoma-like nodules (post-mortem examination by Kolisko). Such decided regenerative changes, though rare, have been repeatedly observed in cases of acute (or "subacute") atrophy of the liver not connected with syphilis, and on the whole it is best to retain the term "acute" atrophy, unless the clinical course is decidedly subacute.

¹ An interesting question arising is whether this hypothetical toxin (produced in the body by the spirochaetes or otherwise) has a more or less specific cytotoxic action on the hepatic secretory cells analogous to the more or less specific (haemolytic) action of the toxins on the red blood-corpuscles in cases of haemoglobinuria (? blackwater fever, paroxysmal haemoglobinuria, &c.), or whether the liver in cases like the present one is selected merely as a "locus minoris resistentiae."

² Doubtless the normal liver, like other organs, is endowed with a considerable reserve of functional power. Progressive destruction of the hepatic cells is tolerated by the organism until the "breaking point" is reached. Then symptoms of "icterus gravis" suddenly arise and are usually quickly followed by death.

I am indebted to Mr. S. G. Shattock and Dr. Ledingham for kind assistance in the microscopic examination of these cases and to Dr. Rolleston for several references to the literature of "focal" and other necrosis of the liver, and I have especially to thank my colleague Dr. Fürth for his great courtesy in handing over to me the material of the second case.

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A Report upon the Pathological Condition of the Aorta of King Menephtah, traditionally regarded as the Pharaoh of the Exodus.

By S. G. SHATTOCK.

THIS interesting relic was sent to the Royal College of Surgeons by Dr. G. Elliot Smith, with the permission of Professor G. Maspero, Directeur-général du Service des Antiquités de l'Égypte. The mummy is reputed upon an Alexandrian tradition to be the Pharaoh of the Exodus. It was found in 1898 by M. Loret in the tomb of Amenhotep at Biban-el-Muluk, Thebes, and was brought to the Museum at Cairo in 1900. The mummy was unwrapped in July, 1907, by Dr. G. Elliot Smith (acting on the instructions of M. Maspero), who has furnished a report upon it in the *Annales du Service des Antiquités de l'Égypte*, 1907. It was found to be that of an old, almost bald man; the thyroid cartilage was ossified. The embalming wound lay in front of the iliac crest and parallel with Poupart's ligament. All the viscera had been removed except the heart. The skin was thickly encrusted with chloride of sodium; the body had been packed with a white cheesy material of the kind which Professor W. A. Schmidt considered to consist of the decomposition-products of a mixture of butter and soda.

As the interest attaching to the specimen is almost exclusively historical, I may give one or two citations from Dr. G. Elliot Smith's report:—

"The mummy of this Pharaoh was found in 1898 by M. Loret in the tomb of Amenhotep II at Biban el-Muluk, Thebes, and was brought to the Museum in Cairo in 1900. In his *Guide du Visiteur au Musée du Caire*, M. Maspero makes the following remarks: 'Momie du Pharaon Ménéphtah, fils et successeur de Ramsès II, trouvée dans le cercueil de Setnakhtî. M. Loret crut y reconnaître la momie du Pharaon hérétique de la XVIII^e dynastie, Khouniatonou. M. Groff affirma le premier que c'était Ménéphtah, et la lecture du cartouche, tracé en écriture hiératique sur la poitrine de la momie, démontra la justesse de son opinion. Le fait était d'autant plus intéressant à constater que Ménéphtah serait, d'après une tradition d'époque alexandrine, le Pharaon de l'Exode, celui qui, dit-on, aurait péri dans la mer Rouge.'

"Even without the evidence of the writing on the shroud many details of the process of mummification would have enabled us to put this mummy into the same group as those of Ramses II¹ (unrolled by M. Maspero in 1886) and Siptah and Seti II (unrolled by me in 1905); and the physical characters of the mummy itself are such as to suggest a near affinity to Ramses II and Seti I. On these grounds there can be little doubt as to the correctness of the identification of this mummy as Menephtah.

"The mummy was wrapped in a sheet of fine linen, which covered the front and sides of the body, but not the back. It passed over the head and extended behind the neck; at the other end it enclosed the feet and ended behind the ankles, its two lower corners being drawn forward and tied in front of the ankle-joint. The name was written in ink on this sheet in hieratic characters across the chest. It was very much faded.

"All the viscera were removed from the body-cavity, except (possibly) the heart. I was able to recognize part of the heart pushed far up into the thorax, but still attached to the aorta. Whether or not it was intended to leave the whole heart in the body, as the practice was in the time of the twenty-first dynasty, I cannot say.

"The aorta was affected with severe atheromatous disease, large calcified patches being distinctly visible.

"The body had been packed with that white cheesy material, such

as I found in many mummies of the priests of Amen (of the twenty-first dynasty). My colleague, Professor W. A. Schmidt, considered the material (in the case of the latter mummies) to consist of the decomposition-products of a mixture of butter and soda.

"A very curious feature of this mummy is the complete absence of the scrotum, which was certainly removed before the process of embalming was complete. The penis was, however, left intact.

"The hands were placed in the position of grasping sceptres, each 15 mm. in diameter, the thumbs being in the position represented in the bas-reliefs.

"The skin of the body is thickly encrusted with salt, which my colleague, Mr. W. M. Colles, has examined and found to be sodium chloride."

The piece of aorta sent to the College is 3 cm. in length. On gently brushing away the debris from the surface there was exposed a perfectly well-defined arterial wall, which, although readily torn, was of firm glue-like consistence, and of a deep brown colour. The material was not dry, but moist, as though impregnated with some hygroscopic material. This condition is attributable to the chloride of sodium and the mixture of butter and soda used in the embalming; the friable material on the aorta was distinctly salt to the taste. As deduced from the microscopic sections afterwards prepared, the debris removed consisted chiefly of the outer, more fibrous coat, or adventitia, the preservation of the rest of the wall being due to the large amount of elastic tissue in its composition. At one of the cut ends the section has passed through a slightly curved calcareous plate 6 mm. in diameter, which lies in the deeper part of the internal and muscular coat, its inner surface being still covered with a thin layer of the intima. At the other end the cross-section of the arterial wall is of uniform and not abnormal thickness, and presents no disease that is obvious to the naked eye. The general form of the vessel is throughout regular, without any abnormal dilatation.

A small portion of the material from the apparently unaltered end, after being placed for some days in absolute alcohol, was soaked in water for forty-eight hours, and then in solution of gum arabic; the sections were prepared in the usual way by means of the freezing microtome; they were readily cut, and their subsequent manipulation offered no difficulties. After being transferred to distilled water, some were stained with Ehrlich's hæmatoxylin, followed by eosin; others with carbol thionine (two minutes, washed at once in absolute alcohol, cleared with

clove oil and mounted in xylol balsam), and some were examined in Farrant's medium without being stained at all. Certain of the unstained sections were first immersed for forty-eight hours in 20 per cent. acetic acid, and others in 10 per cent. hydrochloric acid.

EXAMINATION OF THE UNSTAINED SECTIONS MOUNTED IN FARRANT'S MEDIUM.

These sections display a series of long, parallel, wavy lamellæ of elastic tissue, disposed at regular intervals. Between them there is an intervening material of greater breadth than the lamellæ themselves, and in this there lie groups of minute refractive particles which want the regular spherical form of fat, and are evidently of inorganic nature. The intervening material, where not infiltrated with calcareous particles, exhibits distinct traces of a subdivision disposed transversely with respect to the lumen of the vessel, parallel with the sections of the elastic lamellæ, and representative of the muscle-cells. In other places the material between the lamellæ consists chiefly of a close network of fine elastic fibrillæ (*see* fig. 1, p. 126).

The section comprises only the middle coat, the presence of the inorganic particles in the interlamellar substance indicating the calcification of the muscle-cells, and the whole picture accurately reproducing that presented in senile calcification of the media of the aorta. That this is the lesion present is shown by the action of dilute acids. Did the particles in question consist of sodium chloride they would have disappeared during the passage of the sections through distilled water.

SECTIONS TREATED FOR TWENTY-FOUR HOURS WITH 10 PER CENT. HYDROCHLORIC ACID AND EXAMINED IN GLYCERINE.

The general structure is unaltered; the elastic lamellæ are unaffected. The crystals have disappeared from the intermediate substance. After treatment for twenty-four hours with 20 per cent. acetic acid the crystals similarly disappear; the elastic lamellæ and the fine elastic fibrillæ of the intervening substance remain unchanged.¹ When the

¹ In sections of the normal aorta the muscle-cells of the media are comparatively few, and by no means constitute the whole of the interlamellar material; the rest, as told by the use of Unna's acid orcein stain, consists of a delicate network of fine elastic fibrillæ.

action of 10 per cent. hydrochloric acid is watched upon an uncovered section on a slide, the excess of water having been first removed, no liberation of gas takes place, showing that the inorganic material contains no calcium carbonate.

That the particles do not consist of calcium oleate, resulting from the combination of calcium salts of the plasma with oleic acid set free by the

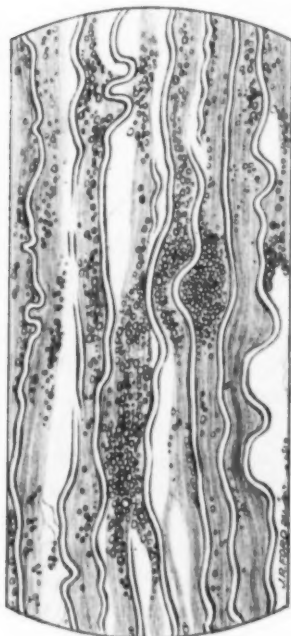


FIG. 1.

Fig. 1.—A section of the aorta, as studied in Farrant's medium, after having been first transferred to distilled water. The normal series of parallel elastic lamellae of the media are preserved in an unaltered condition. Extensive groups of particles of calcium phosphate are deposited in the interlamellar muscular substance. ($\frac{1}{8}$ obj.; oc. 4.)

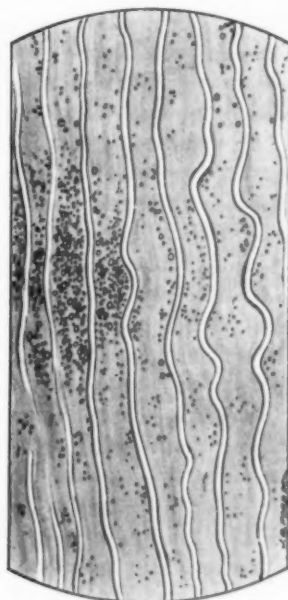


FIG. 2.

Fig. 2.—A section of the aorta of a man aged 79, fixed in formol. The section was prepared in the same way as the preceding. It shows a similar deposition in the interlamellar tissue of the media. ($\frac{1}{8}$ obj.; oc. 4.)

splitting of fat in a degenerate media, is proved by the absence of oil droplets (oleic acid) in the sections treated with 10 per cent. hydrochloric

acid. When oleate of lime is observed microscopically during the addition of 10 per cent. hydrochloric acid the solid particles immediately give place to fine oily droplets of oleic acid. Nothing of the kind occurs in the sections.

EHRlich's HÆMOTOXYLIN AND EOSIN.

The elastic lamellæ take a faint pink coloration in sections so stained and afterwards dehydrated with absolute alcohol, cleared in clove oil, and mounted in xylol balsam. The chief colour of the section is due to the staining of the intervening material. This is of a somewhat pale reddish violet, but no cell-nuclei are brought out in the tissue. There is a clear indication of a striation parallel with the elastic lamellæ and evidently resulting from the disposition of the muscle-cells.

CARBOL THIONINE.

Sections stained for two minutes, washed in absolute alcohol, cleared with clove oil, and mounted in xylol balsam. The elastic lamellæ are stained of a pale sky blue; the intervening material is of a duller, more violet tint; the latter material is abundantly infiltrated with small particles of calcium phosphate.

A second series of sections cut from another piece of the aorta have a further interest, inasmuch as they show remnants of the deeper portion of the intima. This is distinguished by the greater fineness and closer disposition of the elastic lamellæ which chiefly compose it. In the sections stained with carbol thionine the structure is very distinctly brought out, the fine elastic lamellæ and fibres being coloured of a pale blue. The area of the intima included in the section presents nothing abnormal.

On the Behaviour of the Nucleoli in the Cells of Malignant Growths.

By C. E. WALKER and GEORGE DEBAISIEUX.¹

OBSERVATIONS made in this laboratory upon the migration of the nucleoli in the cells of various animals and plants suggested the investigations here described into the behaviour of the nucleoli of the cells of malignant growths. It has been demonstrated that in the normal cells of many organisms the nucleoli multiply within the nucleus, generally by budding, less frequently by a process similar to the division of a single drop of viscous fluid into two drops. Two or more nucleoli—sometimes as many as six or seven—may be observed in some cells of animals and plants. Nucleoli seem often to be thrown out of the nucleus, the nuclear membrane re-forming with great rapidity, frequently leaving a crater-like depression in it. The staining reaction of the nucleolus changes after it has left the nucleus. Sometimes the nucleolus seems to be disintegrated in the cytoplasm; more rarely it passes out of the cell altogether. This phenomenon has been observed in the cells of the following organisms: *Hydra fusca*, *Spongilla fluviatilis*, *Phascolus* (bean root), large and small intestines of rabbit, Planarian worms and normal human skin. The phenomenon is essentially the same in all these organisms, differing only in detail. It is interpreted as being connected with metabolism taking place within the nucleus, and a transference of the products from it into the cytoplasm.²

The observations here described show some considerable divergence from what is apparently the course of events in the case of the nucleoli in the cells of normal tissues. The materials used were chiefly epithelioma and cancer of the breast in the human subject, and a spheroidal-celled carcinoma occurring in mice, given to the laboratory by Professor Ehrlich. For purposes of comparison the cells of the skin and of adenoma of the breast have also been examined. The material

¹ From the Cancer Research Laboratories, University of Liverpool.

² "On the Multiplication and Migration of Nucleoli in Nerve-Cells of Mammals." By W. Page May and C. E. Walker. *Quart. Journ. Exper. Phys.*, No. 2, 1908. "Observations on the Nucleoli in the Cells of *Hydra Fusca*." By C. E. Walker and Alice L. Embleton. *Ibid.*, No. 3, 1908. Observations on the nucleoli in the other organisms mentioned by C. E. Walker and Frances Tozer will appear shortly in the same journal.

used has generally been preserved in Flemming's fluid (strong formula), and most of the preparations have been stained with a triple stain consisting of basic fuchsin or safranin, methylene blue, and Unna's orange tannin. This method of staining is very suitable for the study of the true nucleoli which are stained differentially.

OBSERVATIONS.

The nucleolus dealt with here is the "true nucleolus." In normal cells it is a body generally spherical in shape, occasionally oval, bounded by a definite membrane, the contents being usually homogeneous or finely granular in structure.¹

There are great differences in the number and shape of the nucleoli among the cells of the same malignant growth. Though, as has already been said, the nucleoli in normal cells are generally spherical or oval, in the cells of malignant growths they are very frequently quite irregular in shape (figs. 2, 3, and 7). In the normal cells of the breast or skin there are generally only one or two nucleoli. In the cells of cancer one frequently finds five or even seven in the same nucleus. The nuclei of cancer-cells are frequently multilobular, and in these cases nucleoli are generally found in each lobule (fig. 1). Perhaps the most striking feature of the nucleoli in cancer-cells is that they are less compact and less homogeneous than in the normal tissues. Not only are they more numerous, but their form is irregular, and one finds in the nucleus several irregular masses which can only be distinguished from the chromatin by their volume and staining reaction.

The nucleoli found in normal cells are often observed to have small masses of chromatin lying upon their surface. This is also found in the case of the nucleoli in cancer-cells. Irregularity in the shape of the nucleoli is not confined entirely to the cells of malignant growths. We have found it also in the cells of inflammatory tissue, but not nearly to such a marked extent as we have found it in cancer-cells. In no tissue have we found so many irregularly shaped nucleoli as in malignant tumours, and we have never found them in the corresponding normal tissue.

In so far as the division of the nucleoli is concerned, our observations upon cancer and epithelioma cells generally enables us to confirm in all points the observations published from this laboratory with regard to

¹ See Wilson, "The Cell in Development and Inheritance," p. 34. Walker, "The Essentials of Cytology," pp. 12 and 13.

this phenomenon upon nerve ganglion cells, the cells of hydra, and various other cells, and in certain plant-cells. The first stage in the division of the nucleolus is generally a small excrescence upon its surface (fig. 7). This excrescence grows in volume and travels away from the nucleolus from which it arose, the two bodies being joined by a fine filament (fig. 4). In some cases the two bodies do not separate at first, but remain in contact. In a very few cases we have seen the division occur by a process similar to the division of a drop of viscous fluid into two drops (fig. 5). Occasionally we find nucleoli budding after they have left the nucleus (fig. 11).

The migration of the nucleoli from the nucleus into the cytoplasm is often difficult to find. There are several reasons for this: It has already been said that the nucleoli are often irregular and fragmentary, and it may well be that the migration, instead of taking place *en bloc*, may be carried out by the migration of small isolated masses. Besides this, cells or portions of cells are frequently found engulfed in the cytoplasm or the cells of malignant growths, and some of these may stain in a manner that is more or less similar to the staining of the nucleoli. In the greater number of these cases, however, a careful examination shows whether a particular body is a nucleolus or an inclusion. Sometimes the nucleoli are carried out of the nucleus by the edge of the razor. In such cases one finds the nucleus membrane is torn, and it is easy to recognize the place from which it came. Besides this, by always carefully orientating the sections on the slide it has been easy to avoid this cause of error.

The process of migration takes place as follows: The nucleolus is generally at first situated more or less in the centre of the nucleus. It approaches the nuclear membrane, and causes a protrusion at this point. It then passes through the membrane (fig. 8), the membrane being re-formed behind it very rapidly (fig. 9). Sometimes the nucleolus remains in contact with the nuclear membrane for some time, but more often it migrates to a certain distance in the cytoplasm (figs. 10 and 11).

The change in the staining reaction in the nucleolus after it leaves the nucleus is not nearly so marked in the case of the cells of malignant growths as has been described in the case of nerve ganglion cells. Generally nothing more happens than that the nucleoli in the cytoplasm are stained a pale pink, while those within the nucleus are stained purple or bright red.

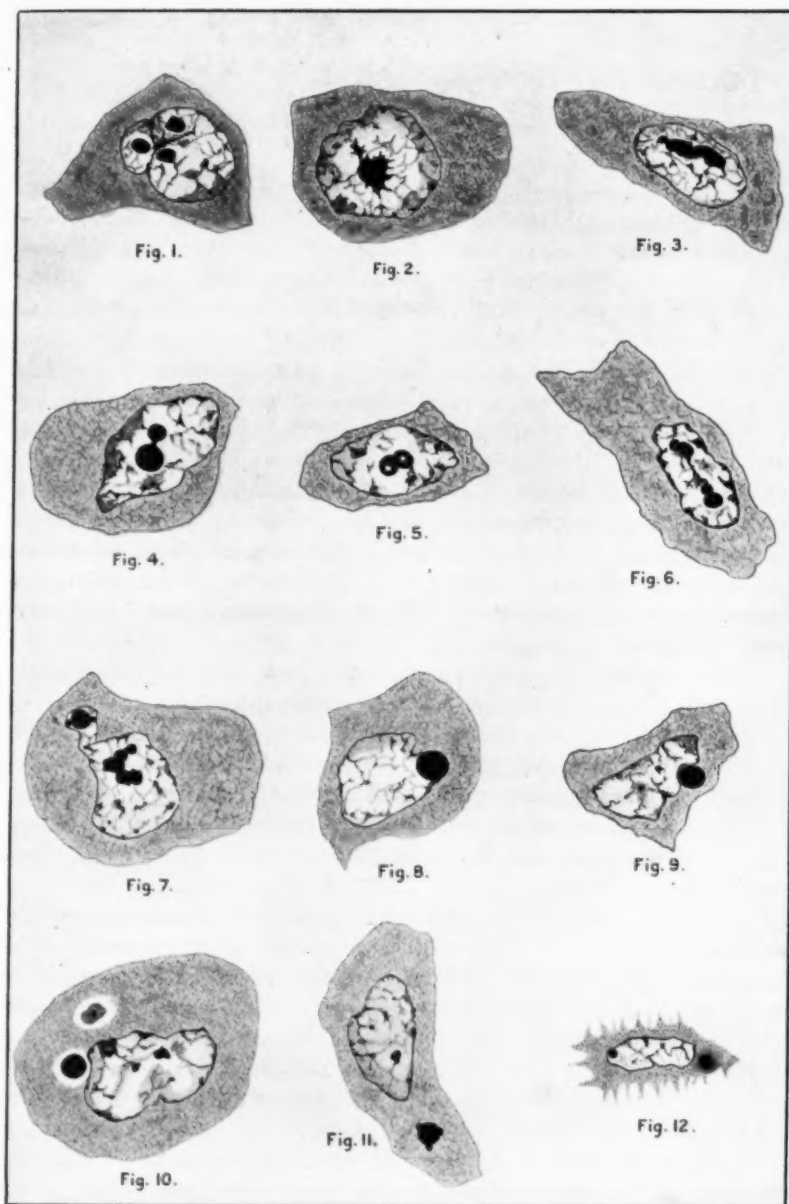
CONCLUSIONS.

As these observations are concerned only with cells in the vegetative condition, and do not apply in any way to cells in process of division, it seems probable, as has been suggested in the case of normal cells, that the multiplication and migration of the nucleoli is intimately connected with and dependent upon metabolism taking place in the nucleus. The cells of malignant growths divide more frequently, and therefore multiply much more rapidly, than the cells in normal adult animal tissues, and this implies rapidity of growth, and may account for the differences between the nucleoli and their behaviour in the cells of malignant growths and those in the cells forming normal tissues. Although some malignant tumours are comparatively slow growing, it must be remembered that in the adult mammalian organism the cells forming the various organs and tissues do not under normal conditions multiply except for purposes of repair. Thus in the skin and epithelial lining of the alimentary canal we find more divisions than elsewhere. Two exceptions to this are the interstitial cells of the testis, which continually produce spermatozoa, and the blood-producing tissues (*e.g.*, bone marrow). In the slowest growing of malignant tumours there must be more multiplication of cells than usually takes place in the normal adult tissues (with the above-mentioned exceptions). It is necessary to point out also that the absence of mitotic figures in a malignant growth does not necessarily mean that the cells are not multiplying. Amitotic division is very frequent in malignant growths, more common than in any normal mammalian cells, excepting, perhaps, leucocytes. The fact that nucleoli actually in process of passing through the nuclear membrane are very rarely found suggests that this stage is a very brief one. In the case of nerve ganglion cells it was found that nuclei that had been extruded from the nucleus often passed out of the cytoplasm in minute portions and not *en bloc*, so it may be that in the case of cancer-cells, where the phenomenon is apparently more frequent, the nucleoli may pass through the nuclear membrane in some similar way on some occasions, and not always *en bloc*, as appears to be the rule in normal cells.

If the conclusion with regard to normal cells be correct, and the multiplication and extrusion of the nucleoli be dependent upon and caused by nuclear metabolism, then these observations suggest that such metabolism is more rapid and greater in the cells of the malignant growths examined than in the cells of the corresponding normal tissues.

DESCRIPTION OF FIGURE.

- FIG. 1.—Cell from carcinoma of breast. Multilobular nucleus with nucleoli in each lobule.
- FIG. 2.—Cell from same growth. Irregular nucleolus.
- FIG. 3.—Carcinoma of mouse. Irregular nucleolus.
- FIG. 4.—Carcinoma of breast. Nucleolus budding. Bud is travelling away from nucleolus.
- FIG. 5.—Carcinoma of cervix uteri. Nucleolus dividing. Uncommon form of division.
- FIG. 6.—Same growth. Two nucleoli both budding.
- FIG. 7.—Carcinoma of breast. Irregular nucleolus in centre of nucleus. A nucleolus in small lobule at upper part of nucleus shows two buds in a very early stage.
- FIG. 8.—Carcinoma of breast. Nucleolus passing out of nucleus.
- FIG. 9.—Same growth. Nucleolus just out of nucleus. Membrane is re-formed.
- FIG. 10.—Same growth. Nucleolus in cytoplasm.
- FIG. 11.—Same growth. Nucleolus budding.
- FIG. 12.—Normal epithelial cell from skin.



Behaviour of nucleoli in the cells of malignant growths.

**Dilatation of the Ureter without Obvious Mechanical
Obstruction (Idiopathic Dilatation).**

By CHARLES R. BOX.

It is a matter of common knowledge that there is considerable difficulty in accounting for many cases of hydronephrosis on the obstruction theory, and the mode of production of the dilatation of the renal pelvis has led to much ingenious speculation. The hydronephrotic condition may occur alone, or be associated with dilatation of the ureter, but the ureter is rarely, if ever, found dilated without the occurrence of a similar change in the pelvis of the kidney. Both hydronephrosis and dilatation of the ureter may occur without either hypertrophy or dilatation of the urinary bladder. The observations of Mr. Shattock on idiopathic dilatation of the bladder and the speculations he has entered into as to its nature have induced me to record briefly two instances of unilateral dilatation of the whole ureter which have come under my observation, and in which no definite obstruction could be found.

The first instance was in an emaciated male infant, aged 3 months. The left kidney was somewhat larger than the right, and the left ureter dilated, the right ureter being considerably less in calibre. The pelvis of the left kidney was also moderately enlarged and communicated with the ureter by an orifice which readily admitted an ordinary probe. The dilated ureter suddenly narrowed to ordinary proportions where it passed through the coats of the bladder; its vesical orifice was not obstructed.

In the second case the ureter was chronically inflamed as well as dilated. The infant, aged 6 months, came under observation on account of vomiting and a slight syncopic attack. The urine, which was acid, contained albumin and pus-cells as well as a few granular and hyaline tube-casts. A rounded swelling, which varied in size, was detected in the left loin. The child succumbed, and, post mortem, the left ureter was found dilated to thrice the size of the right. The pelvis of the kidney also shared in the dilatation. The mucous membrane of both the ureter and the renal pelvis was thickened, granular and discoloured. The vesical orifices of the ureters were patent and of the

same size. Microscopically the affected kidney showed evidence of moderate interstitial nephritis.

Although both of these patients were male children, the facts that the dilatation was unilateral in each, and the bladder not hypertrophied, exclude the possibility of the condition being due to phimosis or urethral obstruction. In the second case the definite signs of chronic inflammation suggest that the dilatation may have been the result of pyelitis and ureteritis. It is conceivable that acute inflammatory swelling of the mucosa might be sufficient to temporarily block the narrow extremities of the ureter, especially if spasm were superadded. In the first case, however, no sign of inflammation and no mechanical obstruction of any kind could be demonstrated.

In a tube like the ureter, whose contents are passed onwards by a continuous succession of peristaltic waves, the abolition of contraction over a limited segment might conceivably be sufficient to produce obstruction.

According to Foster's "Physiology" there is reason to believe that the rhythmic contractions are neither evoked in consequence of mechanical distension of the ureter by the urine, nor in response to chemical stimulation of its interior by the urinary constituents, although the force and frequency of the contractions are in some way determined by the activity with which the urine is secreted. The contractions appear to arise spontaneously in the wall of the ureter, possibly after the initiation in the renal pelvis, much in the same way that cardiac contractions arise in the muscular tissue of the heart. Like the heart the ureter may exhibit rhythmic contractions after removal from the body. Other authors (*e.g.*, Tigerstedt) merely state that contractions of the ureters appear to be started by the entrance of urine into them.

If Foster's conclusions are entirely correct, it is difficult to see how the theory of abolition of reflex action, owing to hypæsthesia of the mucous membrane, which Mr. Shattock has advanced as a possible explanation of certain cases of dilatation of the urinary bladder, can be applied to explain "idiopathic" dilatation of the ureter. But the phenomena of renal colic appear to indicate that the ureter is, after all, in some way supplied with sensory nerves, although it yet remains to be proved conclusively that afferent impulses, arising in the mucous membrane, initiate the contractions which drive on the urine.

A Preliminary Note on the Examination of the Fat in the Liver in Health and Disease.

By E. L. KENNAWAY and J. B. LEATHES.

FOR many reasons special interest attaches to the combinations of fatty acids found in the liver. As a general rule the amount of fatty acids present in this organ is found to be comparable with that found in the heart or kidneys, and it is often less. But under exceptional circumstances in animals that are apparently normal, and not only in those that are diseased, the amount is excessive and then may be very much greater than the largest amounts ever found in the heart or kidney. In pathological conditions in man 60 per cent. and 70 per cent. of the dry solid matter of the liver may be fat, and Rosenfeld has found such amounts too in dogs that have been treated with phlorizin in starvation. There is no other organ in the body in which anything like such large amounts of fat ever occur. Moreover, as was shown by Hartley and Mavrogordato,¹ when these large quantities of fat occur in the liver the fatty acids are not those which normally characterize the fats and lipoids of the liver and other viscera, highly unsaturated acids with an iodine value of about 130, but consist largely of the saturated acids and oleic acid, and therefore absorb in extreme cases hardly more iodine than the acids that can be obtained from the fat of adipose tissue. This and other facts suggest, as has been pointed out before, that the liver stands in a different relation to the fats of the body from all other organs, and that fat which is put into circulation from the connective tissues undergoes some preliminary treatment in this organ before it is distributed to the other viscera in which it is finally oxidized completely.² This preliminary treatment would seem to consist in part of changes in the fatty acids whereby they are converted into those less saturated acids which are characteristic of the fat in the working organs, but which are generally absent from fat stored in adipose tissue. Since it has been repeatedly found that lecithin and other phosphatide lipoids from various sources yield on saponification fatty acids with a

¹ P. Hartley and A. Mavrogordato, *Journ. Path. and Bact.*, 1908, xii, p. 371.

² Leathes, "Problems in Animal Metabolism," 1906, p. 118.

high iodine value, it is possible that this change in the character of the fatty acids occurs only after the acids have been built up into these more complex combinations. It is known that phosphatide fats are present in the liver in considerable amounts; they constitute frequently a half, or perhaps more than a half, of the fat that the organ contains. And it may therefore be a special function of the liver to build up out of the connective tissue fat which reaches it, in the first instance lecithin and the related lipid substances, for the use not only of its own cells but it may be for those in other organs as well, and the desaturation of the acids may occur only after they have entered the lecithin molecule and have come within the sphere of influence of the phosphoric acid and other groups which the lipoids contain, but which ordinary fats do not. One of the first points which suggest themselves for investigation, therefore, is a comparison of the nature of the fatty acids that occur in the simple glycerides present in a normal liver, with that of the acids occurring in combination with phosphorus and nitrogen. The present communication contains the results of some preliminary experiments on these lines, which form part of a study of the functions of the liver in relation to the metabolism of fat in health and disease.

In the first place it was necessary to effect some separation of the different combinations of fatty acids present in the liver. For this purpose the livers were minced and treated with alcohol; the alcohol was strained off after twenty-four hours and the solid matter pressed in a screw press. This process having been repeated several times the combined alcoholic extracts were evaporated *in vacuo* at a temperature not above 50° C. while a slow stream of carbonic acid passed through the fluid. In some cases the extraction with alcohol was followed by treatment with ether, which was then evaporated in the same way. The oily residue left after complete removal of the alcohol or ether was taken up in dry ether and the solution filtered. By the addition of two or three volumes of acetone the first separation is carried out; the acetone precipitating the lecithin and other phosphatides. The acetone solution evaporated in the same way as the original extracts leaves a residue consisting for the most part of simple glycerides with some cholesterin and a little unprecipitated phosphatide. This residue treated with dry acetone dissolves for the most part, and the filtered solution evaporated to dryness leaves a residue soluble in petroleum ether, in which solvent it is best kept for examination. The acetone precipitate dissolved in ether and filtered should be reprecipitated with

acetone repeatedly till everything soluble in acetone is removed. The final ether solution is then treated with two or three volumes of absolute alcohol, when a precipitate is obtained in which jecorin will be present, while lecithin remains in solution. The alcoholic solution evaporated down leaves a residue which is taken up in absolute alcohol and may be kept dissolved in that solvent or else in petroleum ether. The alcohol precipitate reprecipitated from ether by means of alcohol and washed with alcohol can if protected from moisture be preserved as a dry powder.

In this way the substances soluble in ether are divided into three fractions: (1) a fraction composed mainly of simple glycerides with some cholesterin, soluble in acetone; (2) a fraction soluble in alcohol which may be referred to as the lecithin fraction; and (3) a fraction insoluble in alcohol, the jecorin fraction.

The three fractions obtained in this way from the liver of pigs (two series), from a dog, and from a goat have been examined as follows: a weighed amount, about 1 gm., of each of the fractions was treated with potash and alcohol on the water bath for one to two hours. The soap solution obtained was washed with hot water into a Liebermann flask, made acid with sulphuric acid and shaken with a known volume of petroleum ether; and after the separation of the two fluids had taken place a fraction of the petroleum ether solution removed, evaporated to dryness, at first in a stream of carbonic acid and then *in vacuo* over the water bath. The weight of the residue gives the percentage of fatty acid, contaminated it may be with some cholesterin, contained in the sample. The weighed fatty acids serve then for a determination of the iodine value.

The acid aqueous fluid left in the Liebermann flask after removal of the petroleum ether can be used for determination of nitrogen and phosphorus, only in that case any precipitate that there may be, insoluble both in aqueous acid and in petroleum, must be oxidized too, since it contains both nitrogen and phosphorus. During the saponification small quantities of nitrogen escape as ammonia. This was caught in titrated sulphuric acid, the heating with alkali being conducted in a stream of hydrogen. This procedure, though complicated, presents certain advantages over the more direct combustion of separate portions of the original fatty materials in Kjeldahl flasks which, owing to the large amounts of fatty acids contained in the material analysed, requires care and long heating. In some cases the latter method was employed, in most the former.

In Table I are given, for comparison with our results in Table II: (1) The percentage of fatty acids and the iodine value of these acids obtained from the adipose tissue fat of the species of animals to which the data in Table II apply; (2) figures relating to the composition of lecithin of the theoretical formula containing two stearyl radicles, to that of a lecithin obtained by Erlandsen from the bullock's heart muscle, and to that of three preparations obtained by Baskoff from the liver of the horse; and (3) similar figures for the preparations of jecorin analysed by Drechsel and by Baskoff.

The figures given in Table II, the results of our work on the lines described above, show, as we expected, that we were seldom if ever dealing with completely isolated chemically pure substances, but they show that a separation has been effected which is sufficient for the solution of the question propounded in the early part of this communication. The preparations soluble in acetone, referred to in the first group of columns of the table, were, to judge from the percentage of fatty acids which they contained, much nearer to simple glycerides than to lecithin or jecorin. But since the extracts of the liver may have contained free fatty acids, and certainly would contain some cholesterin, and the presence of these in any considerable amount would tend to raise the figure given in this column for the percentage of fatty acids obtained on saponification by the methods used, a truer indication of the degree to which the simple glycerides of fatty acids have been separated from complex lipoids in these preparations is given by the nitrogen and phosphorus content. This shows that they cannot have contained, at any rate, more than from 10 per cent. to 20 per cent. of lecithin or jecorin. And, therefore, the iodine values of the preparations in the first column show clearly that the highly unsaturated acids in the liver are not confined to the phosphatides. For instance, if the iodine value of 120 obtained in the preparation from the pig's liver No. 1 were due to a mixture consisting of 20 per cent. of fatty acids from lipoids and 80 per cent. of fatty acids such as occur in the adipose tissue of pigs, with the iodine value 55, then the iodine value of the acids mixed with these latter would have to be 380, which is so much greater than that found for the fatty acids obtained from the other fractions, that it is certain that the simple glycerides occurring in the pig's liver, if they have come from the animal's adipose tissue, must have been changed in character and been converted into acids which are less saturated. The highly unsaturated acids, then, are not confined in the liver to the compounds containing phosphorus. These latter compounds, however, to judge

from the figures in the table, contain acids which, generally speaking, are more unsaturated than those in the form of simple glycerides. This would be the result to expect if fatty acids when they arrive in the liver are of the type that occurs in the adipose tissue and, generally speaking, in the food, and then in the liver they undergo a process of desaturation; so that the preparations dealt with in the first column represent fat in process of being desaturated, partly still unchanged, partly already changed, whereas the fatty acids in the second and third columns are in a later stage more completely desaturated and further combined up with phosphoric acid and other groups into the lipid molecules of "lecithin" and "jecorin."

The iodine values are, it must be noted, in all cases minimal values. That this is so is shown, for instance, by the following facts:—

(1) As a general rule, as soon as a liver was received for examination a portion of the fresh-pulped tissue was heated with potash and alcohol in order to estimate the total amount of fatty acids present in all combinations, and the iodine value of the mixed total acids so obtained was determined. Among the human livers, for instance, No. 1 contained 3.45 per cent. of its weight of higher fatty acids with the iodine value 135.6; No. 2 contained 3.2 per cent. with the iodine value 134.5. The mixed acids from all sources obtained in this way had been exposed to a much less prolonged treatment than those obtained from the different preparations of the several kinds of fat, glyceride, lecithin, or jecorin, after they had been separated from one another. In the case of the former the iodine value was determined within forty-eight hours of removal from the body; before this could be done in the case of the latter three weeks or more had elapsed. Accordingly the former—*i.e.*, the mixed acids—have a higher iodine value not only than those from the fat soluble in acetone but also than those obtained from the lecithin fraction. This was done, too, in the case of the liver of pig No. 2. The preliminary examination gave in this case 3.15 per cent. of higher fatty acids with the iodine value of 123.1, which is higher than that found subsequently for the acids from the isolated lecithin fraction, and less only than that for the jecorin fraction, which accounted for not more than, at the outside, 5 per cent. of the fatty acids present in the liver.

(2) But even the iodine value obtained for acids prepared by saponifying fresh liver tissue is lower than the real value, for the heating with potash and alcohol causes some changes in the acids. Thus a preparation of "lecithin" was treated with Wijs's iodine solution in the usual way for the determination of the iodine value and absorbed 83.1

per cent. At the same time another portion of the same preparation was saponified and the percentage of fatty acids contained in it determined: 60·5 per cent. were found and their iodine value was now 120·7. If the unsaponified lecithin had the iodine value 83·1, and contained 60·5 per cent. of fatty acids, these acids must have had the iodine value 137·3. This value had fallen to 120·7 during saponification.

The iodine values given in the table are therefore minimal values, and the argument as to the desaturation of the fatty acids present in the form of glycerides is stronger even than the figures show.

Other points brought out in the table that seem to call for comment even at this stage of the inquiry are the following:—

The lecithin fraction gives figures which do not agree with those for lecithin of the usually supposed constitution. The percentage of fatty acids was always less than required by the accepted formula, as was also the phosphorus; the nitrogen, on the other hand, was higher. Four preparations from the pig's liver No. 1 were analysed: those from the second alcoholic extract, from the third, from the fourth and fifth mixed, and fourthly from a preparation obtained by mixing the lecithin fractions of all eight extractions and reprecipitating this mixture three times with acetone. The percentage of fatty acids in these preparations, omitting the analysis of that from the third extraction which was technically faulty, was remarkably constant, ranging from 62·2 to 63·8. The ordinary formula for lecithin requires about from 68 per cent. to 71 per cent., according to whether palmitic, stearic, or oleic acid is supposed to be present. The figures obtained from the liver of pig No. 2 were lower still; so, too, were those from the dog's liver. Baskoff from the liver of the horse obtained lecithin containing 62·5 per cent. and 63·6 per cent. of fatty acids. The percentage of nitrogen found by him was 2·7, higher still than ours, and that of phosphorus, 3·5 per cent., though higher than our figures, is lower than that required by the accepted formula—3·8 per cent.

The constancy in a number of preparations of these variations from the accepted figures for lecithin may be interpreted as meaning that the lecithin in the liver has a different composition, or, as is perhaps more likely, that the mode of separation adopted fails to separate it from some other phosphatide as yet unknown. The figures for the lecithin from the liver of pig No. 2, showing a still greater departure from the true lecithin figures, lower percentage of fatty acids, lower phosphorus, and higher nitrogen content, would in that case mean that this unknown phosphatide was relatively more abundant in this liver.

The jecorin fraction gives the test for jecorin, reduces cupric oxide, gives a red colour with silver and ammonia, and after hydrolysis an osazone melting at 203° C. The fatty acids present in the preparations from the pigs' livers were fairly constant in amount: ten estimations were carried out on different samples from pig No. 1, two of them being by a different method; three were technically faulty; the seven others ranged between 57.7 per cent. and 53.6 per cent. Those carried out on the final preparations obtained by mixing all the earlier ones and reprecipitating once with acetone and once with alcohol gave 54.3 per cent. and 54.4 per cent. The preparation from pig No. 2 gave the figure 55.0.

The figures given by Baskoff for preparations of jecorin from the liver of the horse are 40.75 per cent. (p. 410) and 37.85 per cent. But since our preparations of jecorin fractions from the dog and the goat give again very different figures in each case, it seems reasonable to expect to find that substances of quite different constitution appear in this fraction according to the species of animal from which it is obtained.

The iodine value of the fatty acids, as pointed out before, is necessarily an understatement of the degree to which they are unsaturated. The preparations from the fourth and fifth and the sixth and seventh extracts of the livers from which the figures in the table marked pig No. 1 were obtained, after being mixed and twice reprecipitated, yielded acids with an iodine value of 133, which at an earlier stage had absorbed from 140 per cent. to 150 per cent. of iodine. And even at this earlier stage the preparations had undergone much manipulation of the same kind as that which resulted in this change. But the high figures given by these preparations seem to show clearly that the most highly unsaturated acids are found in this fraction.

The nitrogen and phosphorus estimations carried out on different preparations from the pigs' livers agree fairly among themselves. The nitrogen figures are lower than those given by Drechsel and by Baskoff for the jecorin from the horse's liver. The phosphorus figures fall within the limits of variation in Drechsel's analyses. The single preparations from the dog and the goat require confirmation, but seem to show differences from the jecorin of the pig or horse like the percentage of fatty acids.

TABLE I.

SIMPLE GLYCERIDES FROM ADIPOSE TISSUE.

	In the	Contain fatty acids, per cent.	Having the iodine value
Dog	...	about 95	...
Pig	...	"	...
Goat	...	"	...
Man	...	"	...

LECITHINS.

	Fatty acids per cent.	Having the iodine value	Nitrogen per cent.	Phosphorus per cent.
Theory for distearyl-lecithin	70.4	—	1.73	3.84
Erlandsen ¹ (bullock's heart)	70.5	110	1.79	3.95
Baskoff ² (horse's liver) (1)	—	—	2.70	3.50
" " (2)	—	—	1.95	4.00
" " (3)	—	—	3.90	3.40

JECORIN (HORSE'S LIVER).

	Fatty acids per cent.	Nitrogen per cent.	Phosphorus per cent.
Drechsel	—	2.86	3.2 to 3.7
Baskoff ²	{ 40.75 37.85	2.52 to 3.1	2.8 " 3.0

¹ Erlandsen: *Zeitschrift für Physiologische Chemie*, 1907, li, p. 71.² Baskoff: *Ibid.*, 1908, lvii, p. 395. The different preparations of lecithin analysed by Baskoff were obtained (1) from the alcoholic extract of the liver; (2) from the ethereal extract; and (3) from an alcoholic extract obtained after extraction with ether.

TABLE II.—SUBSTANCES EXTRACTED FROM THE LIVER BY ALCOHOL (SOLUBLE IN ETHER).

Soluble in Acetone					Insoluble in Acetone				
Simple glycerides (+ cholesterol)					Soluble in alcohol; lecithin fraction				
Fatty acids per cent.	Iodine value	Nitrogen per cent.	Phosphorus per cent.	Fatty acids per cent.	Iodine value	Nitrogen per cent.	Phosphorus per cent.	Fatty acids per cent.	Insoluble in absolute alcohol; jecotin fraction
Dog	84.8 ± 0.2	97.0 ± 0.1	0.69 ± 0.02	0.53 ± 0.025	57.2 (2)	105.5	2.02 ± 2.03	3.35	35.4
Pig (1)	{ 88.4 88.6	116.1 120.1	0.31 ± 0.01	0.61 ± 0.01	62.2 ± 1.0 (3)	125.7 ± 0.3	2.06 ± 0.00 1.92	3.25 ± 0.08 3.08 ± 0.09	(4 and 5) 55.5 57.7
	{ 90.1 92.7	114.9 106.0	0.35 ± 0.01		(4 and 5) 63.8 Mixed and reprecipitated three times	130.0 ± 2.0	[2.52]	3.19 ± 0.04	(6 and 7) 56.0
" (2)	{ 96.2 95.0	75.8 77.6	0.36 ± 0.00	0.23 ± 0.01	57.3 56.5	106.1 108.6	2.40 ± 2.41	3.76 ± 0.14	{ 53.6 53.8 Mixed and reprecipitated twice
Goat	{ 94.2 92.4	87.5 90.0	0.37 ± 0.02	0.14 ± 0.01	57.1 58.8 60.4 57.0	116.8 117.0 96.1 115.1		3.04 ± 3.10	{ 54.3 54.4 55.5
Man (1)	87.5	123.4			57.1	116.8			{ 19.46 104.0 103.6
" (2)	88.1	110.5			58.8	117.0			
" (3)	(a) 89.4	101.9			60.4	96.1			
" (c)	(c) 37.1	69.6			57.0	115.1			
" (d)	(d) 96.2	89.9			56.3	71.6			
" (4)	{ 95.0	89.9							54.2 [110.0]
									48.8

Of the human livers No. 1 was from a case of diabetes mellitus, No. 2 from a case of carcinoma of the breast; neither of these was "fatty," and the latter was free from carcinomatous deposits. No. 3 was from a case of hemorrhagic pancreatitis; it contained 8.54 per cent. of fatty acids, with the mean iodine value 85.7; it was extracted four times with alcohol; this extract (a) contained 18.8 per cent. of fatty acids in forms soluble in acetone and 6.6 in forms that were insoluble in acetone, of which 5.3 per cent. were in forms soluble in alcohol and 1.3 insoluble. This liver was then extracted twice with ether, and this extract (c) was almost entirely soluble in acetone and contained 83.5 per cent. of fatty acids. No. 4 was from a case of mitral disease and was also fatty, containing 8.1 per cent. of fatty acids with the iodine value 87.9.

Pathological Section.

February 16, 1909.

Mr. S. G. SHATTOCK, President of the Section, in the Chair.

A Statistical View of the Opsonic Index.¹

By M. GREENWOOD, Jun.

I OWE the Section an apology for accepting my friend Dr. Grünbaum's invitation to speak upon a subject my knowledge of which is slight. An explanation of, if not an excuse for, this presumption is the fact that for some time past my colleague, Dr. Douglas White, and myself have been attempting to throw light upon the accuracy of opsonic determinations with respect to the tubercle bacillus by the aid of methods not widely used in pathological laboratories. No one is more conscious than ourselves that our work is incomplete and leaves untouched many problems of the first importance. Nevertheless, the results of our survey may not be altogether without interest and may enable others to attain some measure of certainty in a matter of practical importance and theoretical interest.

I desire to offer a further apology for the form my remarks will take. I shall, perhaps, seem to explain at length processes which might either be held to require no explanation or, on the other hand, to be essentially *arcana* of statistical science, and as such to be accepted or rejected without discussion by non-statisticians. My excuse is that the methods of a statistician are not well understood by all pathologists, and that I have no ambition to play the part either of a "wit among lords" or of a "lord among wits." Either the principles underlying our work can be intelligibly described and form the basis of a discussion here, or the whole thing is vanity of vanities.

¹ From the London Hospital Statistical Laboratory.

It is, I think, clear enough that the value of opsonic determinations has been disputed by experimental workers whose records appear—at least to a statistical onlooker—to entitle them to some more conclusive answer than the facile charge of imperfect technique. Be this as it may, some vindications of the opsonic method exhibit traces of circular reasoning. Thus, if it be asserted (1) the opsonic content of normal serum is variable, (2) the opsonic index is not an accurate measure of the opsonic content of the serum; a list, however long, of “healthy” people whose indices in terms of one another are close to unity will only refute (1) if (2) be assumed false, and will only disprove (2) if (1) is assumed to be incorrect. Evidently both assertions cannot be simultaneously disproved by one set of determinations without a *petitio principii*. I am not suggesting that the fallacy is ever presented in this crude form, but it does seem to me that a good deal of evidence ostensibly advanced to prove the trustworthiness of the opsonic index does in fact assume the truth of what is to be demonstrated, and that it is necessary to go behind the index, the ratio of two quantities, to the elements from which it is fashioned.

The pivot upon which the whole question turns is evidently the degree of credence to be attached to mean phagocytic power as determined from a small number of cells. We need to examine with some attention the possible sources of error or uncertainty which may be shown to exist. It is not difficult to see that the errors involved fall into two groups. We have first the errors of technique, using the term in its widest sense, and secondly the error dependent on the fact that we measure not all the cells but a sample of them. An illustration will make this clearer. If we desire to obtain a measure of the stature of a race we shall measure the heights of a sample of the adult population. The mean of our determinations will be liable to error owing to want of skill in using the tape or inaccuracy in the latter's graduation; this is the experimental or observational error. The work will also be liable to error owing to the fact that the sample may not typify the characters of the population from which it is drawn. This latter uncertainty cannot be wholly avoided, however expert the observer or however accurate his tape.

In the illustration I have chosen, the separation of the two classes of error is comparatively simple. In the case before us the difficulty is greater. Thus the second error—that which is inherent in any such process—is, speaking in the most general terms, a function of the variation observed in the population and in the sample. But it is

possible that this variation to some, perhaps limited, extent depends upon the technique employed. In other words, the experimental error is not to be wholly isolated from what I shall speak of as the "error of random sampling." For reasons, some of which will be subsequently detailed, I believe the importance of this interplay has been exaggerated. One supposed consequence can be shown at once to have no validity.

It has been argued that since we cannot definitely separate the two causes of error, the method is not ripe for statistical treatment. The rejoinder seems, to me at least, overwhelming. One takes the data furnished by an experienced observer, analyses them, and obtains a measure of uncertainty—*i.e.*, finds that two samples differing one from another by not more than an assigned amount cannot with certainty be referred to separate classes or "populations." How much of this uncertainty depends on the worker's incomplete technical skill, and how much on inherent biological variability, it may not be possible to define. But if the technique of the worker be the best available at the time, it is clear that any *Spielraum* of error found in his work must give the minimum total error of the method at the stage of perfection marked by the epoch to which the measurements refer.

If anyone chooses to say that as the years go by the technique will improve, that measurements made in 1908, or conclusions based on such measurements, will not necessarily apply to the method as practised in 1918, the statistician can only answer that we must deal not with evidence still to be accumulated, but with what we have. We require some provisional test of the value of what is now being done. If in 1909 the method is so fluid as not to be ripe for even provisional testing, it is also not fit to be used as a practical criterion. I must therefore be understood to speak only of the error attaching to the method as practised by the best workers at the present time, and will describe as briefly as I can the principles upon which the study of this error seems to be possible.

An answer to the question, What degree of credibility attaches to the result of a limited past experience used as a basis for judgment? is the goal of thousands of researches within the field of the calculus of probabilities. However these researches may differ in externals, this will be found to be the real object of most. Indeed, it is not difficult to see that Pascal's "Problem of Points," generally taken as the *terminus ab quo* of modern probability work, falls under this category [10]. Attention was not, however, focussed on the accuracy of samples of experiments or observations until experimental science ceased to be a Cinderella of the learned world.

The full treatment of the problem of errors of observations dates from Laplace, whose work was extended by Poisson and, in a sense, completed by Gauss. Very little reflection is enough to convince one that the quantitative treatment of such a problem, its symbolic expression, requires as a preliminary the fulfilment, or assumed fulfilment, of certain conditions. Evidently variation may be conceived to result from many causes; decision between conflicting claims is not generally possible. All Laplace and Gauss could do was to select the most plausible hypothesis and assume its truth as a basis for a provisional treatment of the problem. In effect, the assumptions were as follows:—

- (1) Variation is due to an indefinitely great number of cause-groups.
- (2) These cause-groups are independent one of another.
- (3) Each group taken by itself only contributes a small amount to the total variation.

On this basis, with the help of reasoning admitted by all competent judges to be sound, was constructed the famous "Normal Curve of Errors." I think you will agree that the fundamental axioms of the "normal" treatment of errors are not necessary canons of thought, but working hypotheses. Therefore, if it be found that any given set of observations or measurements exhibits variation not adequately described by a frequency curve of the "normal" type, it is possible that the case is one in which the causes of variation are not capable of being brought within the four corners of the Gauss-Laplace postulates. In other words, the "normal" law requires to be experimentally tested.

Now the history of the matter is very instructive. It was found that many examples of variation were well described by the "normal" curve; that other cases not at first sight to be reconciled with it could be analysed into "normal" components. These experimental results, together with the respect entertained for Gauss and Laplace as scientific leaders, combined to transform their provisional hypothesis into a dogma. All examples of non-"normal" variation were taken to require not an explanation, but an apology, as being in some manner heretical.

The first writer, so far as I know, to doubt the all-sufficiency of the "normal" system was G. T. Fechner [1], an illustrious name in the history of experimental psychology. The influence of tradition was, however, so potent even with Fechner that he did little more than propose an extension—as most statisticians would agree, an illegitimate extension—of the Gaussian method [2]. Indeed, it will be found to this day that many German statisticians, while explicitly repudiating the generality of the "normal" system, implicitly adopt

processes which *look* as little like the Laplace-Gaussian method as possible, and *are* in fact hampered by most of its restrictions. Karl Pearson was the first writer who, dropping the "normal" conditions, proposed frequency systems which included the "normal" curve as a particular case, and could be put to the test of experiment [7].

We are not here concerned with the mathematical form which has been given to these generalized error-curves; the point I am trying to make is an experimental one. If a system of measurements varying round a mean value presents itself in the course of experimental work, it may be that an adequate description of it will be furnished by the "normal" curve. It may be that the "normal" curve fails, and every other frequency system, those of Pearson included, also fails. In such a case the experimenter might either conclude that the system is not homogeneous or that it is an example of variation hitherto uncatalogued. If, however, while the "normal" curve fails one of the Pearson curves describes the observations with great exactitude, then we are justified in refusing to adopt the measurements of the limit of error deduced from the postulates of the "normal" system, and must frame other tests in accordance with the hypotheses upon which the Pearson type curves are founded.

Agreeably to these fundamental principles, Dr. White and myself have undertaken a tolerably extensive study of the variation observed in the enumerations of phagocytic cells made for the purposes of the opsonic index with respect to the tubercle bacillus. Our material consisted of fifteen counts of cells made by Dr. Alexander Fleming, Dr. T. P. Strangeways, and his co-workers Miss Fitzgerald and Dr. Whiteman [3]. The smallest count in the series was of 400 cells, the largest one of 2,000. The majority included 1,000 cells. We employed the usual processes of statistical analysis, and found that while not one of these counts could by any possibility be regarded as "normal," all, with one unimportant exception, were extraordinarily good examples of variation described by skew curves. The types discovered were those known as Pearson's first and fifth types. The charts exhibited are examples from our series; all tell the same story, so that it is unnecessary to trouble you with a multitude of diagrams. On the analytical features of these curves I do not propose to dwell. Anyone who is interested in the matter will find a detailed examination of them in a recent memoir by Dr. White and myself [4]. Some general features of the results are, however, worth immediate notice. In the first place, it is apparent that the distributions are highly

asymmetrical or, in technical language, exhibit "skewness" of a high order. This, of course, is an experimental proof that the Laplace-Gaussian postulates with reference to the causes of variation are, taken as a whole, inapplicable to these examples of phagocytosis. It is an interesting matter of speculation, at any rate, to inquire how this variation may conceivably have arisen.

Sir Almroth Wright has offered a solution which will, I hope, be examined by some speakers this evening. If I understand him correctly, the suggestion is as follows. In any mixture of serum, corpuscles and bacillary emulsion, the actual number of bacilli present may be large enough to give each phagocyte more bacilli than are ever ingested even by the most highly charged cell in the count. But, since the mixture of the respective constituents is not perfect, all phagocytes have not the same chance of ingesting bacilli; some cells have numerous bacilli within reach, as it were; others are far away from the well-spread board. In other words, variation in bacterial content is not so much a matter of inequality in phagocytic powers as of the cells not having each the same chance to secure prey. The discussion of this hypothesis is outside my sphere. I can only be expected to say whether, if true, it would account for our results. If the hypothesis were true, the Gaussian postulate that no one cause-group contributes much to the total variation does not hold for phagocytic counts. Therefore, a skew distribution might be expected, and would, not improbably, take the form actually observed. Beyond this I must not go.

Accordingly, while such an hypothesis is consistent with our results, it is to be remembered that a multitude of other hypotheses, including one of a true biological variability of the cells, would accord equally well with the ascertained facts. Thus the observation that all our frequency curves have a negative start—allowing for a bacillary content of less than zero per cell—may by some be thought to warrant a belief that certain phagocytes are, if the expression be permissible, negatively chemiotactic. It is further to be observed that even if Sir Almroth Wright's hypothesis find entire acceptance, the necessity of testing the method on the basis of skew frequencies remains. Let it be granted that with perfect mixtures the variation would disappear—an extremely rash assumption in face of the known variability of most biological phenomena—we find even in Dr. Fleming's counts, which represent the best technique available at the present time, well-marked skew variation. As I said at the beginning, we are concerned not with the academic question whether

the method will in the dim future prove highly accurate, but with the practical one—how accurate is it now?

I next pass to a comparison between the curves obtained from Dr. Strangeways's material and those yielded by Dr. Fleming's data. The experimental conditions of the two workers were not the same. Dr. Strangeways and his collaborators, Miss Fitzgerald and Dr. Whiteman, used an emulsion expected to give a frequency of about 1.5 bacilli per cell for a "control" count.¹ Dr. Fleming used a thicker emulsion. For theoretical reasons I had surmised that with a thicker emulsion the skewness would be diminished, and hoped that a sufficient approach to symmetry might be attained to allow of using a "Gaussian" test. Although I was in so far justified that the skewness is distinctly less in Fleming's counts, it is still very marked; so that even were this difference in skewness to be entirely attributable to the thicker emulsion of Fleming's technique, it is plain that under possible experimental conditions the emulsion cannot be made thick enough to render any application of the "normal curve" practicable.

It is interesting to notice that the Fleming series affords direct evidence that the limit of thickness compatible with accurate counting has been reached. In a single count, although the curve was a good fit graphically, it did not give a good fit when arithmetically tested. This was found to be due to one group near the upper end of the scale, and is attributable to the difficulty of saying whether a cell contains five or six bacteria.

In speaking of the reduced skewness of the Fleming counts, I guarded against saying that the thickness of the emulsion was the only factor involved. In my opinion, Dr. Fleming's counts show signs of not being random samples, but selections. The evidence for this is as follows: The correlation between the mean of a distribution and the second moment is known in terms of the third moment, standard deviations of mean and second moment, respectively, and the number of observations. If, therefore, we calculate its value on the basis of the largest count, that of 2,000 cells (by Dr. Strangeways), we can, assuming the regression to be linear, find the probable values of the second moment associated with the mean of each count. In this way we found that while the observed values in the Strangeways series agreed admirably with the calculated ones, this was not the case with the Fleming counts.

¹ I am informed by Dr. Strangeways that the counts from his material which we have analysed were all made by him and his colleagues on slides prepared by an expert in opsonic determinations. This fact evidently deprives critics of any justification for attacks on Dr. Strangeways's experimental technique.

I frankly admit that this evidence is technical, that it is not complete, and that I have no shadow of right to ask you to accept it as a complete proof of my contention. I have brought the matter forward because at least it is fair to point out that such differences as we can find between the data of Dr. Strangeways and of Dr. Fleming are favourable to the former's method of work. The suggestion that Dr. Strangeways's material is unworthy of analysis may be regarded as altogether idle.

I now pass to the question, What bearing have these results on the practical value of the opsonic test? Let us look at the matter entirely from the graphical standpoint. Take any one of the diagrams¹ you please—for instance, that marked T.A. Conceive of it as representing the whole "population" of cells involved in any one case. Then remember that in practice one takes a small handful of fifty or hundred cells blindfold from this "population." The most superficial consideration of the diagram leads at once to the following conclusions:—

(1) We are not equally likely to obtain random samples, each of which differs by the same quantity from the mean of the whole "population," one in excess, the other in defect. In other words, samples are liable to biased error.

(2) The range beyond the mean is greater than the range up to the mean. Therefore, great positive deviations might conceivably occur in random sampling while correspondingly great negative deviations are excluded. This, of course, only applies to extreme deviations.

(3) Owing to the marked skewness of the curve the mean is not a good descriptive constant; the mode (the most frequently occurring as distinct from the mean value) is more reliable.

The truth of proposition (1) and (2) will be further illustrated in the sequel; (3) is, I think, of some importance.

The whole object of employing a mean value is to have expressed by a single constant some of the main features of a distribution. In any symmetrical frequency, such as the "normal" curve, the mean coincides with the mode and is evidently the most useful single constant we can obtain. When, however, there is a marked skewness, as in our phagocytic counts, since the mean and mode are far apart, knowledge of the mean only might lead us to form a very incomplete idea as to the general features of the count. The mode, on the other hand, is relatively better for summarizing purposes, although, of

¹ All the curves fitted are published in Greenwood and White's memoir [4].

course, no one constant will render an adequate account of any frequency-system.

For these practical reasons and certain theoretical ones which I need not discuss, I suggest that it is advisable to replace the mean by the mode for testing indices. Unfortunately, the true mode can only be found by a relatively long arithmetical process; still, even a value determined by inspection or the roughest graphical considerations is not improbably better than the arithmetic mean value. You will notice that this proposal bears a slight resemblance to that of Simon involved in his so-called "percentage" index [8]. The method actually recommended by this observer does not impress me as being very accurate, and his statistical treatment of the data is unsatisfactory, but his work, perhaps, merits more attention than it seems to have attracted in this country.

I have now examined some of the consequences which flow from the existence of skew variation in phagocytic counts. Two of these, the existence of a biased error and its corollary that high and low indices are not equally reliable, will be examined further. In a Gaussian "population" the expression measuring the variability of a mean is accurately known, as also the modifications of method necessary to be employed when the samples are very small [9]. Had the distribution been "normal," the preceding investigation would have been unnecessary, and we could have stated with some confidence that the limits of significance for samples of seventy-five (with the present technique) are about 20 per cent. in either direction [5]. The Gauss-Laplace frequency system does not, however, apply to phagocytic counts, and we have to ask ourselves whether any plan of testing the variability of the means of samples taken from a non-Gaussian "population" can be framed. I am not prepared to say that no such expression can be deduced by strictly algebraic analysis—indeed, Dr. White and I may be able to publish some such theoretical considerations, but at present we have to rely on directly experimental methods.

The largest count in our series was one by Dr. Strangeways and his colleagues of 2,000 cells, a count made up of eighty samples of twenty-five. Taking the eighty means as separate observations, we analysed the resultant frequency distribution and obtained the curve exhibited in the diagram. It is a well-marked example of type 1, sensibly skew and an excellent fit. While admitting that the number of means upon which the result is based is comparatively small, the excellence of the fit leads me to think that it affords a provisional solution of our problem so far as

samples of twenty-five are concerned. As before, imagine that this curve represents the whole "population" of means. Then the chance that drawing a sample from this population will give us a result not differing from the "population" mean by more than an assigned amount is merely a question of areas. Thus the chance in favour of getting a sample mean of 1.353 or less is the ratio of the area from the start bounded by the ordinate 1.353 to the rest of the area. The odds against this are 5.68 to 1. In terms of the "real" mean this deviation corresponds to an index of 0.8. In this way we see that the odds against random sampling being responsible for indices of 0.9, 1.1, 1.3, are respectively 1.88 to 1, 3.6 to 1, 12.46 to 1. This amounts to saying that even so extreme a deviation as 1.3 might be reasonably expected to appear rather less often than once in thirteen times merely as a result of random sampling and without any coincident physiological change in the "population" whatever. In other words, no definite importance can be assigned to variations within the limits of 0.8 and 1.3 at least.

I am, of course, aware that workers seldom rely on samples of twenty-five cells, although very far-reaching conclusions have in the past been based on counts of this size. How far the limits of probable variation would be narrowed by a consideration of samples of fifty or one hundred is not certain. That they would be narrowed to some extent, but not it may be very much, is probable. The belief that a sample of fifty is twice as good as one of twenty-five indicates a somewhat primitive state of knowledge, and I have never been able to ascertain what criterion is adopted by those who assert that while twenty-five cells are useless one hundred give infallible results. My doubts whether the limit of non-significant variation will be much reduced are partly founded on the form of the skew curves and partly, but not so much, on the result that samples of seventy-five, even on an assumption of Gaussian conditions, are subject to an error of some 20 per cent. [6].

Naturally these latter remarks are but surmises which Dr. White and I hope shortly to destroy or confirm by an application of the method just described to much larger samples. In my opinion we have so far succeeded in demonstrating that:—

- (1) Phagocytic distributions are markedly asymmetrical.
- (2) This asymmetry, although reduced, is not removed by emulsions of (from the experimental standpoint) maximal thickness.
- (3) The mode of a phagocytic distribution is a more reliable constant than the mean.
- (4)—A corollary of (1)—Positive and negative deviations will not occur in random sampling equally often.

Further, we have not proved, but rendered somewhat probable, that samples of twenty-five are unreliable, at least within the limits of 0·8 and 1·3. For the reasons above recited I can feel no great degree of confidence that samples of fifty or one hundred will necessarily give results of much greater accuracy.

One last word. I have endeavoured to describe as plainly as I can a matter not free from difficulty and obscurity. If in so doing I have used any expression either dogmatic or over-confident, you will, I earnestly hope, attribute it not to malice aforethought, but to imperfect powers of accurate expression. This must be almost the first time a statistician has had the temerity to address you on a definitely pathological subject. The time will come when such co-operative work is not the exception but the rule, to the benefit of both kinds of investigator. In consideration of the harvest which the future may reasonably be expected to yield, you will overlook the mistakes and crudities of the first unskilful gleaners.

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DISCUSSION.

Colonel LEISHMAN said that his own experience of the opsonic index in the case of tubercle was so small as to be of little value, but of late years he had had considerable experience of the index in relation to immunization with typhoid vaccines. Although this work had been done with normal individuals or normal animals, the results could be contrasted with those obtained in disease, as both were examples of acquired immunity—one natural, the other artificial—and there was no essential difference in the process. His own feeling on the matter was that it was still too early to pronounce definitely upon the limits of significance of the opsonic index. In the typhoid work of his colleagues and himself they did not regard as significant variations of 0.1 and 0.2, as was done in the case of tubercle. In typhoid work ranges much higher were obtained, and they did not attach much significance to anything under 0.5. They had been trying to prepare a vaccine which would give rather more regular and better results than did the typhoid vaccine in the past, and had prepared many experimental vaccines with which men and animals had been immunized. The efficacy of such vaccines had been partly judged by estimations of various protective substances, such as bacteriolysins, agglutinins, &c. As the result of this work at the Royal Army Medical College he and his colleagues had come to place more reliance on the estimations of the phagocytic power than on the estimations of other substances. They were single-hearted in their wish to get an effective vaccine, and if they had found that the method of opsonin estimation was of no value they would have thrown it overboard for one which was more reliable. The recent statistical results with regard to typhoid vaccine bore out the idea that the vaccines which had been found best by the opsonic test would in practice give the greatest amount of protection, and that appeared to him an argument of some value. With regard to the technique of opsonic estimations in general, he would make a few comments. A good control was the basis of every opsonic estimation, and one could not have much confidence in small variations unless one knew the limits of normal variations. In most cases the control serum was derived from one healthy individual, but he thought it ought always to be "pooled"—*i.e.*, the serum of several healthy individuals should be taken and mixed in corresponding volumes, and the test carried out with such pooled serum. That came out strongly in the animal experiments in connexion with typhoid. Since the pooling had been adopted the results had been much more regular and more easy of interpretation. Then he believed it was not fair, at any rate in dealing with diseased bloods, to leave altogether out of account the patient's own corpuscles. Some of their experiments supported this, and he believed some of the President's and Dr. Dudgeon's work pointed to the same conclusion. He thought there was something to be said for the original technique of the method—namely, taking the patient's own blood and merely adding to it the bacteria suspended in some neutral fluid; clotting was not allowed to take place, nor even decalcification by the addition of sodium citrate, but the patient's own cells and plasma were allowed to act on the test

dose of bacteria. He felt sure that the details of this method could be readily improved if it were found to give more reliable results in connexion with tubercle. With regard to the emulsion, Greenwood's earlier work showed that the question of variability might depend, as Sir Almroth Wright had suggested, upon the individual cells not getting equal opportunities of phagocytosis owing to inefficient mixing. Although Dr. Greenwood now found that this did not give a complete explanation of the variability, it was well known to investigators that the evenness and uniform density of the emulsion was a feature of the greatest importance. He thought improvements were possible in that respect, and that more effort should be made to standardize the emulsions used in a series of experiments. A quantity sufficient for the series might be made and then stored and sealed in capillary tubes, each containing sufficient for a single estimation, and turned upside down night and morning to avoid precipitation. Another important point was the strain to be used; whether it should be virulent or non-virulent, one freshly isolated, or one which had been long subcultured. It was only by observing uniformity in these and other points that one could hope to get more consistent results. His view was that the method did give good results, but that too much had been expected of it. This appeared evident from Mr. Greenwood's interesting communication, and he had been much struck with several of the suggestions which he had made in connexion with the skewness of the curves he exhibited. He (Colonel Leishman) had often found that, even with the utmost care, certain cells, even in normal blood, did not ingest any bacteria, while other cells might take up an enormous number; for the latter he suggested the name "glutton-cells." The influence of these upon the index was very great. He had long felt that one would get a fairer idea of the average phagocytic power of a given serum by taking the number of bacteria most frequently ingested. Dr. Greenwood had now pointed out that the curves he had shown gave a similar indication, and that the "mode" might afford a better measure than the mean. He had thought hitherto that the neglect of such glutton-cells would have been a statistical dishonesty, but now he had heard it was not an immoral proceeding he would await with interest the result of work on such a plan. Even with its present limitations he believed the method to be of great value, but he thought it capable of further improvement as a result of careful observation and research. He believed that vaccine therapy could not be expected to give its best results in the absence of opsonic estimations.

Dr. LEDINGHAM said that the Section owed much to Mr. Greenwood for his lucid presentation of the statistical side of the question. Whatever method one adopted of estimating the opsonic content of a serum, one would ultimately have to reckon with this peculiar phagocytic distribution brought out by Mr. Greenwood. Investigators would be glad if Mr. Greenwood would give some ready arithmetical methods of determining the mode without having to chart out the distribution every time. The author had dealt with the accidental errors of the method as actually practised and with the precision to which it was possible to attain. He (Dr. Ledingham) wished to refer to the systematic error of the

method. It was now generally admitted that the opsonic effect was a complex one, being due to the interaction of amboceptor and complement. A technique similar to that employed in the comparison of hæmolytic or bacteriolytic sera had been employed so far only to a very small extent in opsonic work, but the results obtained certainly justified the further elaboration of a method which would take into account the complex nature of the opsonic antibody. Several observers had established that complement-variations occur in normal persons and animals, and it seemed quite reasonable to suppose that many of the minute variations in the opsonic index recorded by certain workers (effect of exercise, menstruation, pregnancy, &c.) really depended on small changes in complement-content.

Mr. GOADBY said that, like Colonel Leishman, he had not done a large amount of estimation of the opsonic index in tubercle; his work had been more with the organisms of the staphylococcic and streptococcic type, and in connexion with organisms found in inflammatory conditions about the jaws. He had treated a number of persons at the National Dental Hospital and elsewhere by means of vaccines. A large number of his indices fell into line with the curve which had been suggested that evening. It was interesting to note that in many cases in practical experience one got more variation in indices above the normal than in the indices below. One fallacy which had not been referred to in connexion with absolute estimation of the index was that in a number of sera one got bacteriolysis of the bacteria in the cells themselves, and that might occur to a great extent if twenty or thirty minutes were left for the ingestion of the bacteria. He was also interested in the curve which showed that the thickness of the emulsion rather tended to reduce the error, because experimentally he had found that he got better results by using a thicker emulsion of bacteria for a shorter time than by thin emulsion. He had used a ten minutes' exposure with a thick emulsion, which had been well centrifugalized. Another point was that which was brought out by Marshall's work, a point foreshadowed by Dr. Ledingham—namely, that the opsonic index plotted on a dilution of serum gave a curve which showed a most distinct variation, which was in direct relationship to the degree of dilution of the serum. As the dilution increased, so the curve fell away; it was a logarithmic variation. And if the estimation of opsonic indices directly made by the observer in that way held such a good mathematical relationship, evidently something in connexion with those estimations was particularly good. Whether such a series of estimations in large numbers would give the same skew curve, he did not know. It looked as if in future the mode was a matter to which attention would have to be directed rather than to the mean. He asked whether Mr. Greenwood could give an easy method of determining the mode instead of the mean. Would it be possible to do it by taking the mode of a given series of estimations by graphically dotting on either side of the mean the number of cells which showed a variation above and below?

Mr. GREENWOOD, in reply, said that he did not see any satisfactory way of determining the mode simply.

Pathological Section.

March 16, 1909.

Mr. S. G. SHATTOCK, President of the Section, in the Chair.

Case of Portal Thrombosis, associated with Stricture of the Urethra and Double Mitral Disease : Fatal Hæmatemesis.

By HENRY CURTIS and T. W. P. LAWRENCE.

THE patient, an English engine-driver on a mine in a part of Rhodesia where malaria is endemic, was aged 52. He had been exposed there to all weathers for twenty years, and was admitted under the care of one of us ostensibly only for retention of urine and the considerable hypogastric pain resulting. He was otherwise apparently in good general health, but was found to have double mitral disease and an impassable stricture of the urethra.

The first attack of retention had occurred in Africa eighteen years previously—in 1887; this, and the passage of instruments necessary, recurred some twelve months later. In 1898, at Salisbury Hospital, Rhodesia, retention again occurred, requiring suprapubic drainage, and, later, internal urethrotomy, a No. 10 silver catheter being tied in. After eight or nine weeks in hospital, the catheter was regularly passed for eighteen months, when, after an attack of malaria, the patient found it necessary to reduce the size of the instrument, proceeding gradually from No. 8 to No. 3. For four months before the present time he had not been able to pass any instrument at all, and, on admission, even after hot baths, morphia suppository, &c., the finest catheter or bougie could not be insinuated. At the operation, subsequently performed, the stricture was confirmed to be practically impermeable. No enlargement of the spleen was detected. The next day, little or nothing escaping

per urethram, nor on suprapubic puncture, it was arranged to do an external urethrotomy, but this had to be postponed, for, two days after admission, the first of the only two rigors noted set in, probably indicating the point of acute exacerbation of the portal infection. At length, after a day or two of normal temperature, thirteen days after admission, with a view to urethrotomy, an attempt to anæsthetize with chloroform was made, but had to be abandoned, probably owing to the double mitral disease. But seven hours later the first hæmatemesis took place, amounting to about 6 oz., and this was followed the next morning by the first intestinal hæmorrhage, quite $\frac{1}{2}$ pint of liquid red blood being mixed with light, clay-coloured, semi-formed stool. The same afternoon, about twenty-four hours after the attempted anæsthetization, the second and only other rigor occurred; 15 oz. of blood-stained urine were passed, neutral or slightly acid in reaction, of specific gravity 1016, and containing a trace of albumin, a further 11 oz. escaping during the night. The amount of urine, almost colourless and remaining of the same character (except for the tinge of blood seen earlier), then gradually increased nearly to the normal. Three days later, under eucaïne, the first stage of an external urethrotomy (Wheelhouse's operation) was performed, the operation being completed two days later, after which urine was freely passed, both through the tied-in catheter and through the perineal wound. Two days after the completed operation there was marked collapse, and on this and the next day diarrhœa, for the first time, was present, constipation having been the rule. Four days after the operation fatal hæmatemesis occurred.

The symptoms of portal thrombosis exhibited in this case were partial suppression of urine, masked by the retention due to an impassable stricture, two rigors, pains in the loins, two attacks of hæmatemesis, an attack of intestinal hæmorrhage, and, towards the end, brief but marked collapse and diarrhœa. The second attack of hæmatemesis was fatal, twenty-three days after admission. The record is of interest in connexion with the pathology of portal thrombosis, especially as, being actually associated with operative measures, it might have been classed with cases of post-operative thrombosis, so well known to occur after abdominal and, even more frequently, pelvic operations.

The post-mortem and microscopical evidence proved, however, that the portal thrombosis resulting in occlusion more or less complete, and fatal by hæmatemesis twenty-three days after admission, preceded by a considerable time the urethrotomy performed under local analgesia, there being found long-standing chronic pylephlebitis.

AUTOPSY.

(Sunday, December 3, 1905, 2 p.m., four and a quarter hours after death.)

There is a tortuous vein in the skin of the abdomen, slightly to the right of the median line, as noted before the first operation.

Larynx: Entrance and interior, normal; in the mid-line, just below notch, bony, and cannot be cut with the knife.

Lungs: Normal, except for black pigmented areas on the surface.

Pericardium: Normal.

Heart: Not dilated. Right auriculo-ventricular orifice dilated, admitting four fingers; left auriculo-ventricular orifice "buttonholed," edges thickened, very nodular; left auricle is dilated above mitral valve orifice, and easily admits four fingers. The condition indicated mitral regurgitation and stenosis. Pulmonary artery and valves not obviously atheromatous.

Heart substance: Right ventricle, thin-walled; left ventricle, hypertrophied but soft. Several hard, almost calcareous, nodules in the lining wall of the aorta at its origin, but the coronary arteries are neither calcified nor obviously obstructed.

Spleen: Of enormous size (fig. 1, A). Capsule greatly thickened, especially at certain spots, where there are huge round plaques of primrose-yellow, about $\frac{1}{8}$ in. thick, confirmed on section to be only thickened capsule, not extending into the substance of the spleen. Section of the spleen: Connective tissue hypertrophied; substance of light claret colour; looks firm, but breaks easily.

Kidneys: Both kidneys are small, each being not larger than diagram (fig. 1, B). Section: Rather pale and fibrous-looking; very tough on attempts to break down; no cysts; pelvis well shown, but not distended. Ureters not distended above or below.

Bladder: Not distended, but shrunken, and of size and shape indicated (fig. 1, c). (It must be remembered that the perineal drainage would in itself account for the collapsed condition of the bladder.) It shows, behind and below, lateral expansions (fig. 1, E, E¹), proving to be sacculi with orifices symmetrically placed just above, and $\frac{1}{8}$ in. externally to the entrance of the ureters. These cavities are large enough to hold the terminal joint of the thumb, the interior of dirty ash-grey-green tint. The muscular wall of the bladder elsewhere is contracted and hypertrophied. The mucous lining is thickened and chronically congested, with large varicose veins and one projecting papilloma, the size of the

terminal joint of the little finger. Otherwise the lining of the bladder is smooth, rugæ not being marked, neck smooth and but little congested.

Urethra: The proximal $2\frac{1}{2}$ in. to 3 in. of the urethra, from the bladder-neck downwards, is considerably dilated, and its surface smooth. There appears to be an old false passage to the left of the median line.

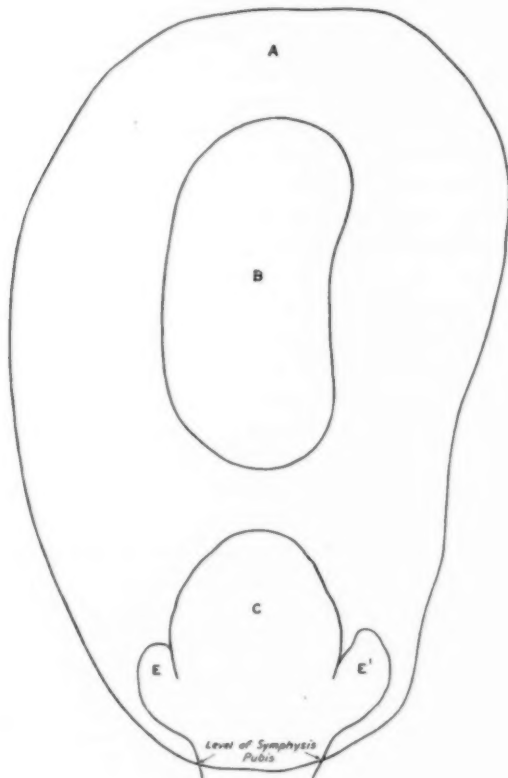


FIG. 1.

Tracing of rough sketch made at autopsy. **A**, spleen (approximately half the natural size, which was about 9 in. by $5\frac{1}{2}$ in.); **B**, shrunken kidney (approximately half the natural size); **C**, collapsed bladder and symmetrical pouches or sacculations (**E**, **E'**).

The stricture is found to have been completely divided at the second stage of the operation (November 29). The roof of the urethra at the

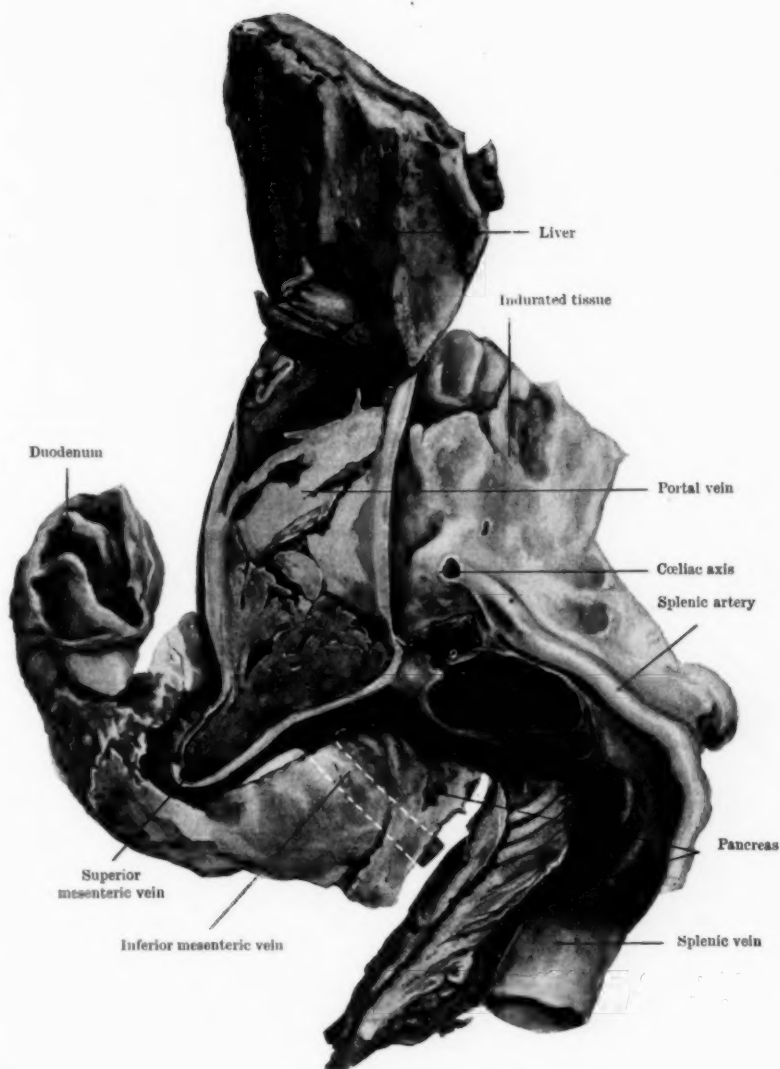


FIG. 2.

Specimen of the portal vein, &c. (From a drawing by W. T. Shiells.)

level of the perineal opening is much scarred, doubtless from previous instrumentation (internal urethrotomy in 1898, &c.).

Prostate: Not enlarged; the mucous membrane lining it is normal and of a light yellow colour. Section: Shows neither lobe enlarged; cut surface white, finely granular, not fibrotic.

Peritoneal cavity: There is no sign of recent peritonitis, and no fluid anywhere.

The intestines, large and small, appear to be quite normal.

Appendix: Normal, small, loosely coiled up; nowhere adherent.

Liver: The right lobe of the liver appears to be forced down below the right hypochondrium, the upper end of the obliquely placed gall-bladder being situated vertically below the right nipple; its apex is directed downwards and inwards towards the umbilicus. The left lobe is greatly lengthened, its long tongue-shaped tip being very firmly adherent to the spleen at the extremity of the left hypochondrium. Surface of the liver: brownish yellow; liver capsule, in one place, close to the lower margin of the anterior surface, near the junction of the right and left lobes, is scarred but not much depressed. Section of the liver: rather paler than the surface, and of brown-yellow-grey or brown-grey tint; granular and fatty, and soft on pressure; the cut surface presents an appearance almost like that of sago-grains, everywhere. Numerous spots of yellow-brown bile in sections of the right lobe of the liver. There are some slight old adhesions beneath the liver, between it and the pancreas (?). A few small lymphatic glands, brownish on surface, are seen in the lesser omentum, soft and brown on section.

Portal vein: The left branch of the portal vein is seen to be much thickened, and externally of opaque straw tint (proved later to be due to great distension with clot of this colour). On trying to remove the liver, a thick band is found passing to the liver in front of the Spigelian lobe from behind the pyloric end of the stomach. This band is as wide as four fingers, and found to lead behind the head of the pancreas; incorporated with the back of this band is seen the portal vein, greatly distended (about three finger-breadths). When this vein is divided there is but little loss of blood, and on removal of the liver nothing but straw-coloured clot is found in the widely dilated left branch of the portal vein, seen in the left lobe of the liver on section. No other vessel is seen affected similarly in sectioning the right lobe of the liver. No pus and no growth in the liver, on the surface or in section.

Pancreas: The head and body of the pancreas is normal on section.

The splenic vein is distended with thrombus, extending from the

spleen the whole way behind the upper border of the pancreas to its junction with the superior mesenteric vein, the inferior mesenteric vein being inserted near the angle of junction of these two vessels, to form the portal vein, here so enormous (about three finger-breadths) as at first sight to suggest an aneurysm, and full of hard old thrombus of pink yellow tint, resembling a mass of yellow resin or beeswax. This extends below into the superior mesenteric vein as far as the lower border of the pancreas. On the surface of the thrombus at one spot, where adherent to the vessel-wall, the thrombus is ash-grey in tint. There is no pus anywhere. The clot is evidently very old, resembling the oldest clot of a healed aneurysm.

The stomach is seen to be distended and a little congested externally. It occupies the entire front of the abdomen, from hepatic to splenic flexures. When slit up it is found to contain more than a pint of blood, partly fluid, but almost more dark clot than liquid. Beyond this and mucus (and slight streaks of white material, either bismuth or faint traces of curdled milk) no other contents are recognizable. The stomach, together with the last one and a quarter inches of the œsophagus and the first inch of the duodenum, being removed, examination of the stomach and duodenum shows there to be no ulceration, old or new, and no petechiæ or other visible evidence in these regions of the source of hæmorrhage (*see later*). There are several distended, slightly enlarged, veins showing through the gastric mucous membrane internally, but the mucous membrane over these is intact. One small papillomatous growth, the size of a pea, is seen in the stomach. The cut surface is pink. There is no infiltration of the deeper coats. Having brought to England the stomach, &c., it has been possible to obtain an independent report of the conditions present, after preservation in a mixture containing formalin and spirit for over three years.

REPORTS BY T. W. P. LAWRENCE.

(1) MACROSCOPIC.

Portal vein, &c.: The portal vein is dilated, its wall thickened, and its lumen almost completely filled with blood-clot. The dilatation is uniform, the diameter of the vessel being 3 cm. ($1\frac{3}{16}$ in.). The thickness of the wall varies from 2 to 3 mm. (about $\frac{1}{8}$ in.) in most parts, but at about the middle of the vein it increases to 7 mm. (a trifle more than $\frac{1}{4}$ in.), and a prominent ridge projects from a considerable part of

the circumference of the vessel into the lumen. The part of the vessel below the ridge is almost completely filled with adherent and partly decolorized clot; over considerable areas, especially of the anterior wall, the clot is not adherent. The clot is firm and is marked by clefts and fissures, especially at its more central parts; it shows no areas of softening. The part of the vessel above the ridge is occupied by pale laminated clot, but the lumen is not so completely obstructed as in the lower part of the vessel, where the blood-stream must have been greatly impeded. The right division of the portal vein is dilated and thickened, and a few areas of calcification, each measuring about 4 mm. in length, are visible on the inner surface. These calcified areas are perfectly smooth, and do not appear to be denuded of endothelium. There is no adherent blood-clot. The clot in the lower part of the portal vein is continued into the terminal 3 or 4 cm. of the superior mesenteric and splenic veins, and apparently completely blocks the latter vessels. The clot in the superior mesenteric vein has the same characters as that in the portal vein, but is not adherent to the wall of the vessel, and probably permitted the passage of a certain amount of blood between it and the vessel-wall; in the splenic vein nearly one-half of the clot as seen in the section is of a red colour, and, although evidently *ante mortem*, has apparently been more recently deposited. In the splenic vein the clot is closely adherent to the vessel-wall. Both of these veins are much dilated (splenic, 1.7 cm. = approximately, $1\frac{1}{16}$ in.; superior mesenteric, 1.5 cm. = approximately, 1 in.), and their walls are slightly thickened. The inferior mesenteric vein is inserted near the angle of junction of the splenic and superior mesenteric veins; it is not enlarged, contains no clot, and its wall is distinctly thickened and rigid. It enters the main vessel at a spot where the clot is non-adherent, and probably, therefore, the blood-stream throughout was not entirely obstructed.

The areolar tissue, situated behind and to the left of the portal vein, in the angle of junction between that vein and the splenic, is indurated. The coeliac axis passes forwards through the most indurated part, but without suffering any diminution in calibre. The portal vein can be separated without difficulty from the indurated tissue, and the contour of the vessel is not altered by any external constrictions. No areas of suppuration are present in the indurated tissue.

The splenic vein is rigid and calcified.

The small piece of liver-substance preserved has the appearance, in section, of being somewhat cirrhotic (but see Microscopic Report).

The portion of pancreas preserved appears to be normal.

The stomach : The stomach is slightly dilated ; the pyloric orifice is narrow but not diseased. The mucous membrane shows no marked changes beyond the presence of a small polypoid projection of the size of a small pea, attached to the summit of one of the rugæ.

The œsophagus : In the lower part of the œsophagus two of the longitudinal folds of mucous membrane are more prominent than normal owing to the presence of dilated veins. The crests of the enlarged rugæ in the lower 3 cm. of their extent are marked by several minute erosions, measuring 2 to 3 mm. in diameter. Two of these are deeper than the rest, and form distinct ulcers with clear-cut edges and with no thickening of their bases or boundaries. The base of each of them is formed by a subjacent vein, and in each case the vein is perforated. The ulceration of these dilated veins at the lower end of the œsophagus appears to have been the source of the fatal hæmatemesis.

(2) MICROSCOPIC.

(Report on Sections of various Tissues.)

Portal vein : Microscopic structure of the portal vein.—There is great thickening of the interstitial tissue in the form of a dense, hyaline, fibrous tissue, poor in cells. The thickening forming the ridge before mentioned is caused chiefly by the organization of adherent blood-clot.

Splenic artery : The splenic artery shows the changes of chronic endarteritis.

Indurated tissue : Microscopic structure of the indurated tissue.—The induration appears to be connected chiefly with the outer coat of the veins, some of which contain granular blood-clot. No new growth is present. The glands in the indurated tissue show some degree of fibrosis, chiefly in the neighbourhood of veins.

Pancreas : The microscopic appearances of the pancreas are normal.

Liver : Microscopic structure of liver.—A piece of liver-substance adjacent to one of the main divisions of the portal vein shows thrombosis of many vessels in the areolar tissue between the vein and the liver substance. The liver is not cirrhotic. The columns of hepatic cells show a tendency to become broken up in most places, the individual cells being isolated from one another. The cells are reduced in size and their angles rounded ; some of them are atrophic or disintegrating. The cell-columns are widely separated, but the spaces are not

occupied by blood or exudation, and the condition may have been due to œdema. In places the veins contain small masses of granular coagulum adhering to the inner wall of the vessels. Scattered collections of leucocytes are present in places.

REMARKS.

We may conclude from the post-mortem and microscopic reports that the fatal hæmatemesis resulted from erosion of varicose veins at the lower end of the œsophagus, secondary to very extensive though not complete portal occlusion, and especially associated with complete occlusion of the splenic vein.

This occlusion of the portal vein resulted from thrombosis of very long standing, which in turn resulted from chronic pylephlebitis. As to the cause of this pylephlebitis, we must note the absence of:—

(a) Cirrhosis of the liver, stated by Langdon-Brown¹ to be the cause of rather more than 47 per cent. of all cases of pylethrombosis at St. Bartholomew's Hospital, London, in thirty-three years, and by far the commonest cause thereof.

(b) Malignant disease of the liver, or in the portal area (more frequently in the latter), given by the same authority as the next commonest cause of pylethrombosis.

(c) Calcareous degeneration—in the sense of patches with their rough edges or spicules projecting through the lumen into the intima. There was no roughening of the intima corresponding with the position of the calcareous plates actually present, nor were any clots adherent there.

That pylephlebitis and some degree of pylethrombosis long antedated admission to the hospital on November is apparent from the record of the naked-eye condition, and the microscopy of the walls of the several veins entering into the portal system, as also by the similar condition of sclerosis present in the splenic artery.

The date of onset of the acute phase appears to be indicated by the rise of temperature to 100·8° F. on November 11, the day after admission, and the rigor of November 12, when the temperature rose to 103·4° F., a condition at first attributed to malaria, but now considered to have been due to portal infection.

The huge size of the spleen was greater than one sees in uncomplicated malaria, large as they are commonly found to be at autopsies in the Tropics.

¹ "On Pylephlebitis," a thesis which gained the Raymond-Horton-Smith Prize at Cambridge, and was reprinted in the *St. Bartholomew's Hospital Reports* (1901), 1902, xxxvii, p. 59.

(d) Relation to the operation: Operative interference certainly had nothing to do with the onset of the disease, as the initial hæmatemesis, indicating, usually, more or less complete occlusion of the splenic vein, occurred on the night of November 23 after the attempt merely to anaesthetize with chloroform had had to be abandoned before any operation had been commenced.

(e) Relation to peritonitic adhesions: In spite of the presence of adhesions—evidence of local chronic peritonitis—independent examination has shown that “the portal vein could be separated without difficulty from the indurated tissue, and the contour of the vessel was not altered by any external constrictions.”

As to the Positive Factors.

(1) Dilatation of vessel-walls: There was marked dilatation of the portal vein and the vessels uniting to form it. This dilatation, resulting from lost elasticity, became more and more marked with increasing size of the contained clot.

(2) Cardiac disease: It is difficult to say how far the double mitral disease present contributed to the pylethrombosis. It would certainly have added to the general venous congestion. Such congestion would be greatly increased on the administration of an anaesthetic. [See below (4).]

(3) Sepsis: Sepsis has come to be regarded more and more as a most important factor in the etiology of this condition. The clinical history of chronic stricture of the urethra, extending over eighteen years, and the condition of the urinary tract noted at the autopsy, indicated a source of sepsis, which was, in our opinion, responsible in great part, if not entirely, for the damage to the walls of the vessels entering into the portal area (pylephlebitis), which led to thrombosis, and a fatal issue by hæmatemesis.

(4) The anaesthetic: The administration of the general anaesthetic alone may, possibly, have played some part in leading to the complete occlusion of the splenic vein, and so ushering in the hæmatemesis ultimately proving fatal. [See above (2).] If so, it would be very interesting to inquire what part, if any, the anaesthetization plays in those cases of thrombosis which, more properly than this, are included under the term “post-operative.”

It is interesting to note that there was never any ascites, nor jaundice, and diarrhoea only occurred on two successive days (December 1 and 2) before death on the morning of December 3.

The clinical features and post-mortem findings having been dealt with, and an expression of our view as to the pathology of the case, we may with advantage enumerate the signs and symptoms which may be considered pathognomonic of pylethrombosis.

Signs and symptoms of portal thrombosis; Rolleston¹ concisely sums these up as follows: "The clinical manifestations of portal thrombosis present a certain amount of variation which may roughly be correlated with the situation and extent of the thrombosis in the portal area. Thus, if the portal vein alone is involved, the most prominent symptom is that of obstruction to the passage of the blood through the liver—viz., ascites, while hæmatemesis may also occur. If the thrombus occludes the proximal end of the splenic vein where it joins the portal vein, enlargement of the spleen and hæmatemesis may be expected. Thrombosis of the mesenteric veins is associated with intestinal obstruction, diarrhoea, melæna, and collapse. Arranged in the order of their frequency and importance, the chief clinical features of portal thrombosis are: Ascites, enlargement of the spleen, gastro-intestinal hæmorrhages, abdominal pain, symptoms of intestinal obstruction, diarrhoea, and manifestations of toxæmia resembling those in the late stages of cirrhosis, and not unlike uræmia. It will be seen that thrombosis of the portal vein may present the aspect of cirrhosis of the liver, of a gastro-intestinal disorder, or may combine the features of these two groups."

The onset of the condition all are agreed is variable, and the more insidious and chronic the occurrence of pylethrombosis the more difficult it is to judge the exact moment at which the acute phase of more or less complete obstruction of the portal system has commenced. In our own case it is possible that the acute phase really occurred prior to admission to hospital, though the previous history afforded no evidence thereof. We have therefore assumed the acute onset to have supervened upon the chronic about November 12, the third day after admission, when there was a rigor, and the temperature rose to 103.4° F., but fell rapidly to normal after (but, perhaps, independently of) the use of hypodermic quinine (10 gr.). The chart from November 12 to December 3, the day of death, clearly shows that the morning temperature only once rose above normal, namely, on November 24, *i.e.*, the morning following the first hæmatemesis; this occurred at midnight, November 23, after an attempt merely to administer chloroform had had to be abandoned. On the morning of November 24, too, there occurred, with the temperature

¹ "Diseases of the Liver," Lond., 1905.

rising to 102.2° F., the only other rigor definitely known in connexion with the case, twelve days' interval elapsing between the two rigors (November 12 to 24). With the hæmatemesis and rigor the pulse-rate per minute, previously ranging between 62 and 88, average 72 to 80, was raised to 100, 120 to 140; the respiration-rate being 20 to 28, generally being 20 to 22 per minute. There was, at times, pain, occasionally quite severe, across the right loin, but otherwise the signs and symptoms of portal thrombosis chiefly consisted of the two attacks of hæmatemesis, one ten days before death, the second on the day of death

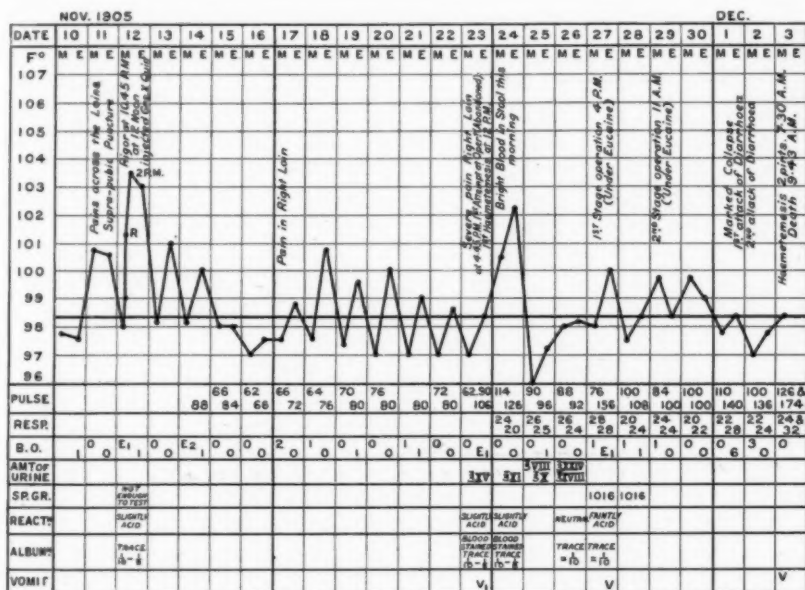


FIG. 3.

—and the immediate cause of it—severe collapse two days previously, associated with two sharp attacks of diarrhoea on that day and the following; some dilatation of a cutaneous vein in the right side of the abdominal parietes. There was no obvious enlargement of the spleen, the huge size of which, indicated in fig. 1, A, came as a “post-mortem surprise,” as frequently has been noted in such cases.

The signs and symptoms actually present were practically almost entirely those due to the splenic occlusion. The non-occlusion of the

condition, was rendered unusually difficult by reason of the existence of such an "impermeable" stricture of the urethra as was demonstrated at the operation, but it is somewhat remarkable, however, that the amount of urine passed increased from little or nothing at first to a little below the normal on the day before the first stage of the urethrotomy (*i.e.*, two days after the second of the two rigors); so that one realizes now that there was some degree of diminution in amount from pylethrombosis, as well as retention from stricture, though the urine passed was not "high-coloured and lithatic," as in pylethrombosis suppression, according to Rolleston, but pale, free from deposit, and of low specific gravity.

DIAGNOSIS.

"This is very difficult," Rolleston says, "and it is only seldom that a correct opinion can be arrived at during life. The sudden onset of ascites, or of hæmatemesis, and their recurrence, accompanied by considerable splenic enlargement, might suggest it. But these symptoms are much the same as those of cirrhosis, with which portal thrombosis is so often associated; it is difficult to differentiate between portal thrombosis and cirrhosis. Cirrhosis is so common, while portal thrombosis is comparatively rare, that in any given case the probabilities are rather in favour of the former, even though the onset of symptoms is sudden and severe. The sudden onset of ascites in a case of cirrhosis led me on one occasion to diagnose pylethrombosis, but the cause was tuberculous peritonitis. In some cases of gastric ulcer profuse gastro-intestinal hæmorrhage with collapse may suggest portal thrombosis. Difficulty is only likely to occur when the gastric ulcer is acute and occurs in an adult male or in a woman who has never had any signs of gastric ulcer, and is considerably past the age at which it is commonly seen. Very profuse hæmatemesis and melæna in a soldier, aged 35, coming on suddenly at Pretoria, and accompanied by a low temperature, led me to an erroneous diagnosis of portal thrombosis; at the autopsy there was acute diphtheritic dysentery, with an ulcer of similar nature in the stomach. In splenic anæmia the spleen is very considerably enlarged, there is anæmia of the chlorotic type, with a diminished number of leucocytes, while recurrent gastro-intestinal hæmorrhages may occur. The disease is essentially chronic, while thrombosis of the portal vein is usually rapid. But in some instances of portal thrombosis, especially when the splenic vein is occluded, the spleen is greatly enlarged, and there may be periodic gastro-intestinal hæmorrhages for many years,

with fairly good health in the intervals. Thus Langdon-Brown reported the case of a woman who had had hæmatemesis at intervals of ten months for twenty years, and in whom the portal and splenic veins were found occluded. Such cases are, however, most exceptional."

Bland-Sutton,¹ speaking of "Obliteration of the Splenic Vein," says: "It is an extremely rare condition, but it is worth considering on account of the peculiar signs associated with it. My experience is limited to one case. A nurse, aged 26, suffered for six years from recurring attacks of profuse hæmatemesis associated with a big spleen. The bleeding was attributed to a gastric ulcer, and the amount of blood she lost in some of the attacks was so great as to reduce the red corpuscles of the blood to 662,000 per c.m. Gradually the corpuscular elements would increase until they reached the proportion of 3,200,000 per c.m. This always indicated that hæmatemesis was imminent and invariably happened within a few days. The woman herself knew when to expect an attack, and eventually died after a profuse bleeding. At the post-mortem examination the splenic vein was large enough to admit the index finger; it contained pouched recesses, and at its junction with the superior mesenteric was blocked by an organized thrombus."

It would be decidedly interesting to learn if this remarkable alteration in the blood-cells has been noted in other cases of thrombosis,² since, if so, its diagnostic value would be so great. [For further remarks on this case, *see* under section on "Treatment."]

PROGNOSIS.

The diagnosis being so beset with difficulties, the practical application of prognosis is very limited, but there is no doubt that the prognosis is infinitely better than in pylephlebitis. From this dictum of Rolleston, our case of mixed phlebitis and thrombosis, one may conclude, was from the first hopeless. Where compensatory circulation can be set up before the obstruction is absolute the patient has a much better chance of surviving, and Langdon-Brown, Osler, Rogers and others have recorded cases where life was prolonged for years (Rolleston). On the other hand, as Langdon-Brown puts it, "it must not be forgotten

¹ *Encycl. Med.*, Edin., 1902, xi, p. 338. (Now *Encycl. and Dict. Med. Surg.*, ix, p. 311.)

² *Addendum*.—Those interested in this subject should consult the article on "Polycythemia, erythrocytosis, and erythræmia," a critical review by Dr. Parkes Weber in the *Quarterly Journal of Medicine*, October, 1908, noting especially Appendix A.

that cases of portal obstruction of some standing may terminate in hæmatemesis (as did our own case), or intestinal hæmorrhage, with a rapidity that is dramatic."

TREATMENT.

The treatment suggested by Rolleston when portal thrombosis is suspected is to determine the coagulability of the blood, and if this and the amount of calcium is found to be increased, 30 gr. doses of citric acid three times a day should be administered, to lessen the excessive coagulability.

Langdon-Brown records a case where the omentum was stitched to the abdominal walls to afford new paths for collateral circulation, as in cases of cirrhosis of the liver. The operation was unsuccessful, and considering the tendency of the blood to undergo clotting in this condition, the perils of extension of the thrombosis must be borne in mind. Possibly this risk might be diminished by administration of citric acid for several days prior to the operation.

In about half the cases portal thrombosis is associated with hæmatemesis or intestinal hæmorrhage. It is therefore of practical importance to consider whether some of the deaths, at any rate from hæmatemesis, can be obviated. Most cases of hæmatemesis occur only when the splenic vein has become blocked by thrombosis, the back pressure resulting from venous oozing, or engorging the probably already varicose gastric, or œsophageal, veins until they give way.

It is clear from Mr. Bland-Sutton's case that such splenic thrombosis may be quite transitory, and may recur frequently. If, as seems evident from the record, the lesion in this case was limited to splenic occlusion, splenectomy, especially with the improved results now obtained, would, we suggest, seem to be the line of treatment indicated in any similar case in future, by allowing ligature and extirpation of the splenic vein. Where indicated by the state of the blood as to excessive coagulability or excess of calcium salts, citric acid should be previously administered as a measure of precaution.

Except in the more chronic cases of portal thrombosis-hæmatemesis, which are rare, the diagnosis of splenic occlusion, like that of portal thrombosis itself, would be far from easy, the chief points to remember being the association of hæmatemesis with a greatly enlarged spleen, which sometimes greatly diminishes after such hæmorrhage, and the alterations in the blood such as were recorded in Mr. Bland-Sutton's

case. Whatever operative measures are contemplated, the risk of the anæsthetic increasing the venous congestion must never be forgotten, spinal analgesia offering, perhaps, the safe solution to the difficulty.

In our own case, pylephlebitis was associated with the pylethrombosis, and would of itself contra-indicate such an operation as splenectomy.

In a case diagnosed to be hæmatemesis from portal thrombosis, in the presumed absence of pylephlebitis, an exploratory laparotomy, with the precautions suggested, might well be undertaken, and if the occluded condition of the splenic vein and the absence of pylephlebitis were confirmed, an attempt might be made to avoid the fatal issue by hæmatemesis by splenectomy.

In the allied condition of intestinal hæmorrhage from thrombosis of the superior mesenteric vessel, the only hope would seem to lie in increasing the collateral circulation: a suitable lateral anastomosis would not only provide for this, but also short circuit the part of the bowel rendered *hors de combat* temporarily by the thrombosis. Unfortunately, this type of case usually runs too acute a course to permit of any effective interference, but though the condition is almost inevitably fatal, we suggest that an appropriate lateral anastomosis (with, or without, in addition, suture of the omentum to the abdominal walls, as in Langdon-Brown's case) is a procedure that should at least be given a trial.

Dr. LANGDON BROWN said that in his figures regarding the occurrence of pylethrombosis to which Mr. Curtis had referred he had been able to find, in 15 per cent. of the cases, that a septic focus was responsible—a fairly high percentage. He thought it likely that sepsis was responsible in the present case. He thought much depended on the intensity of the infection as to whether there was pylethrombosis or whether there was portal pyæmia. If the blood-clots were firm they would probably prevent the septic process spreading further up into the liver, but if the process was intensely infective the clot softened and broke down and the ordinary features of portal pyæmia would ensue.

A Contribution to the Pathology of the Spleen.

By LEONARD S. DUDGEON and W. O. MEEK.¹

THIS work was undertaken with a view to ascertaining the part played by the spleen in certain acute and chronic infective and non-infective diseases. It must be pointed out that this work consists solely of the study of the histological and cytological changes, together with the bacteriological findings in these cases. Special attention was directed to the question of the fate of the erythrocytes in the spleen, in so far as microscopical examination can determine. The cases examined, 87 in number, comprised 46 examples of acute infective and non-infective diseases, 28 cases of diseases presenting a chronic course, and 13 of chronic diseases with an acute terminal infection.

TECHNIQUE.

A portion of each spleen was reserved for microscopy. Sections were cut in paraffin and stained by various methods. Hæmalum and eosin was the routine stain employed. Other methods consisted of van Gieson's, Leishman's, and the Gram-Weigert. In some cases sections were examined for the presence of the free-iron reaction. Cultures were taken from the splenic pulp, as soon after removal from the body as possible, in a large majority of the cases. Films of the splenic juice were made by squeezing a portion of the organ between forceps. The expressed juice was spread on slides.

Leishman's stain was invariably employed for staining the films. A differential count of the cells was made in every case. In most cases 500 white cells were enumerated, but in some as many as 1,000 cells were counted.

The splenic cells were examined for intracellular fat by fixing wet films in formalin vapour, staining with Scharlach R. and counterstaining with hæmalum in over 50 per cent. of the cases. In fourteen instances coarse or fine fat droplets were seen in the cytoplasm of certain of the polymorphonuclear and endothelial cells. The condition was most marked in the cases of acute infection. Fat was never found

¹ From the Pathological Laboratories, St. Thomas's Hospital.

in the lymphocytes, large or small. Sections were stained by the Gram-Weigert method for fibrin in a large majority of the acute cases. Fibrin was found in the splenic pulp or sinuses on only four occasions. These were made up of two cases of pneumonia, one case of epidemic cerebrospinal meningitis, and one of acute septic peritonitis. In the latter case the fibrin-formation was present only immediately beneath the inflamed capsule. In a few other cases a network of fibrin was found confined to the blood-vessels.

An increase of fibrous tissue in the spleen and thickening of the splenic capsule was observed in about one-third of the cases examined, these being mainly instances of long-standing disease. The spleens from cases of cirrhosis of the liver showed fibrotic thickening of the capsule, usually of a marked degree, and a great increase in the amount of fibrous tissue in the interior of the organ. The trabeculae were more numerous and thicker, and the walls of the sinuses greatly thickened.

In all our cases of pernicious anæmia there was fibrosis of a greater or less degree, though thickening of the capsule was found only on one occasion. In cases of long-standing cardiac disease similar changes were seen in a few instances. Thickening of the walls of the arteries was commonly observed, especially in the case of old subjects. In no other groups of cases were these changes seen, except in isolated instances. This increase of fibrous tissue was found on five occasions in the spleens of infants under 3 years of age, the causes of death being respectively sepsis and broncho-pneumonia, measles, acute lymphæmia, epidemic cerebrospinal meningitis and marasmus. In all of those the increase of fibrous tissue was slight. Thrombosis of blood-vessels was noticed several times, and calcification of the vessel-walls in one or two instances. Areas of focal necrosis were present in two cases of typhoid fever.

BACTERIOLOGY.

Film preparations of the splenic juice were made in every instance where cultures were taken, and stained with Leishman's blood-stain. Cultivation experiments were made from a selected area from the interior of the spleen, the surface of which had been rendered sterile in the usual manner. Cultures were taken from more than one region in the spleen whenever it appeared to be advisable. Tubes of suitable media were inoculated according to the nature of the case.

In Group I, which comprises the acute infective and non-infective diseases, a bacteriological examination was made on forty-five occasions,

but in no less than fourteen instances the cultures proved to be sterile, and no organisms were seen in the film preparations of the splenic juice. In four of the cases, however, cultures were sterile, but cocci were seen in the films.

Particular attention must be drawn to certain facts of interest. Six cases of acute pneumonia were investigated in the manner already indicated, but the pneumococcus was cultivated on only one occasion, and in this instance the bacillus of influenza had been isolated from the pus withdrawn from one pleural sac during life. Although the splenic juice was found to be sterile so often, yet cocci were seen in the films in no less than four of the cases. If we contrast these results with those obtained in typhoid fever, we find a striking comparison; out of four cases investigated the *Bacillus typhosus* was cultivated on each occasion, while bacilli were seen in the film preparations in three instances. It cannot be decided whether these differences are dependent upon the fact that the bodies are kept in a freezing chamber in the post-mortem room, and thereby the cultivation of the pneumococcus is rendered a matter of greater difficulty, but we know from experience that the pneumococcus can be grown from the splenic juice in pneumococcus infection in children where the bodies have not been subjected to such a low temperature. Of course, we cannot be certain that the cases of acute pneumonia we are recording were due to the pneumococcus; all we can say is that cocci were seen in film preparations of the splenic juice, that the patients had died from acute lobar pneumonia, and that cultures which were taken were sterile with the one example which grew the pneumococcus. The finding of the *Bacillus typhosus* is only what one would expect, as it is a matter of common knowledge that the bacillus can be readily grown from the spleen at the post-mortem examination on cases of enteric fever. Out of the five cases recorded as acute septicæmia, the *Streptococcus faecalis*, according to the classification of Andrewes and Horder, was obtained twice, and an atypical streptococcus once; in the remaining cases an atypical staphylococcus and a diphtheroid bacillus were isolated. The bacillus of influenza was obtained in one instance from a case of influenzal pyæmia.

In Group II, which comprises the diseases presenting a chronic course, there is little of interest to record. We must refer, however, to the fact that we failed to grow streptococci from the splenic juice in seven fatal cases of pernicious anæmia. In fact, the bacteriological findings in all these cases were strikingly lacking in interest.

In Group III the chronic diseases with acute terminal infection are recorded, and out of this number reference must be made to the bacteriological findings in three cases of endo-pericarditis and one case of pernicious anæmia fatal from acute pneumonia. In the last-mentioned disease the *Streptococcus pyogenes* was isolated from the spleen and also from the heart blood; this case affords a striking contrast to the other cases of pernicious anæmia previously referred to. From the three cases of endo-pericarditis the *Streptococcus pyogenes* was isolated once, the *Streptococcus faecalis* once, and the *Staphylococcus aureus* once.

ENLARGEMENT OF THE SPLEEN.

In about one-half of the spleens examined the viscus was enlarged. This enlargement was due to a variety of causes. Disregarding those causes which may be termed accidental—e.g., infarction, macroscopic areas of necrosis, large areas of hæmorrhage and areas of acute inflammation due to the presence of micro-organisms—the two most evident changes in the large spleens were: (a) The presence of an abnormal quantity of blood, either in the sinuses or elsewhere; (b) a thickening of the walls of the sinuses. In a majority of the spleens from cases dying from acute infection or toxæmia the increase in the amount of blood was marked. The splenic sinuses appeared to be distended with red blood-cells, and in some cases the blood was not confined to the sinuses, but diffused throughout the splenic pulp. As is mentioned elsewhere in this paper, the red cells which fill the sinuses are commonly in process of hæmolysis and disintegration.

In our series of cases the most striking examples of this distension of the sinuses were found in cases of typhoid fever, some cases of pneumonia, septicæmia and pyæmia, toxæmic jaundice, and pernicious anæmia. In all our cases (8) of pernicious anæmia, the spleen showed some enlargement. In one case it weighed 36 oz. The distribution of the blood in this case was remarkable. There was a large amount of pigment present, and the cells lining the walls of the sinuses contained numerous pigment granules which served to mark the limits of these spaces, while the sinuses themselves were practically empty. The splenic pulp was packed with an enormous number of large, ill-staining red cells in process of destruction. The other main cause of general splenic enlargement—namely, fibrosis—was well seen in certain cases of chronic disease, more especially hepatic cirrhosis. Ten cases of this disease were examined, and in all in which the hepatic cirrhosis

was well marked, a practically identical condition was found in the spleens. This consisted in a great thickening of the sinus-walls, due to a deposit of young fibrous tissue. While this fibrosis is evident throughout the sections of these spleens, it is especially well marked close to the endothelial lining of the sinuses. From this cause the sinuses may remain widely patent, though the spleen be almost empty of blood. Sections of such spleens give an excellent picture of the vascular network of the organ. It is scarcely necessary to mention that in a number of cases fibrosis and distension of the sinuses with blood were co-existent.



FIG. 1.

A portion of the splenic pulp from a case of hepatic cirrhosis, showing great thickening of the walls of the sinuses due to a deposit of fibrous tissue. (Zeiss objective, D.D.)

THE MALPIGHIAN BODIES.

During the microscopical examination of the sections the condition of the Malpighian bodies was investigated. The special points noted were their number and size, the presence or absence of "germ-centres," and the occurrence of cellular or other tissue-changes in and around them. With regard to number and size, allowance was made for the fact that in many instances the spleens examined were considerably

enlarged either by the presence of blood in the sinuses or by the great increase of fibrous tissue in the splenic pulp. These factors, by separating the Malpighian bodies, tended to give a false impression of their number.

In the whole series of cases the condition of these bodies appears to be markedly dependent upon the age of the patients. In seventeen cases from children of 5 years of age and under they were poorly marked in only two, while in the majority of instances they were prominent, and in nine showed large "germ-centres" consisting of large cells. Out of the sixteen cases between the ages of 5 and 20 they were well marked or

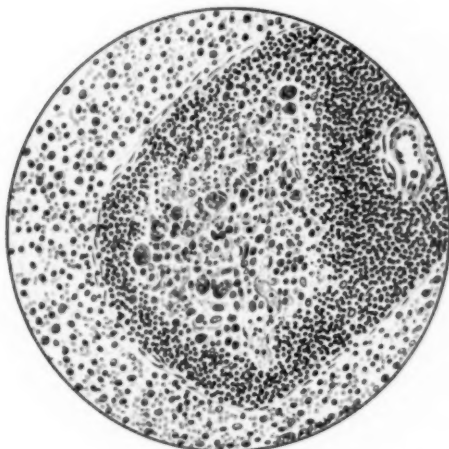


FIG. 2.

A Malpighian body in the spleen, showing nuclear fragmentation at its centre, and phagocytosis of the nuclear fragments by endothelial cells. From a case of acute infective disease in a young child. (Zeiss objective, D.D.)

increased in size in seven instances; in the remainder they were reduced in size. In this series large "germ-centres" were only seen on one occasion—in a spleen from a case of thyroid enlargement with general lymphatic hyperplasia. In cases over 20 years of age the Malpighian bodies in two-thirds of the cases showed a distinct diminution in both number and size. "Germ-centres" were recognized on only two or three occasions. In the spleen from a case of streptococcal septicæmia in a man aged 23 the Malpighian bodies were very prominent with

large centres. In those cases where "germ-centres" were recognized the cells forming them varied in type. In some they were of the small lymphoid group; in others they were larger and endothelial in character. Mitotic figures were seen in these endothelial cells on several occasions. In a few spleens from cases of acute disease nuclear fragmentation and disintegration of the lymphocytes in the Malpighian bodies was noticed, while the large cells in the centres had engulfed lymphocytes and their remains. Occasionally pigment was seen in the Malpighian bodies. Other changes noted were thickening of the central artery, a deposit of fine fibrous tissue in and around the Malpighian bodies, and of pink granular material at their centres. Amyloid change was seen in a case of lymphadenoma. In a case of marasmus in an infant with marked atrophy and fibrosis of the thymus the Malpighian bodies in the spleen were of normal size and showed prominent germ-centres with mitotic figures in the cells.

CYTOLOGICAL CHANGES.

(a) *Finely Granular Polymorphonuclear Cells.*—In the majority of instances of acute infectious diseases these cells, as one would expect, were present in excessive numbers in film preparations of the splenic juice. We may take, for example, seven cases of acute pneumonia; here the average percentage of this variety of cell was 22 per cent. The only instance amongst this series in which there was no excess of these cells occurred in a case of pneumonia complicated by an empyema from which the bacillus of influenza was obtained in pure culture during life. In this case the lymphocytes numbered 70 per cent. and the endothelials 34 per cent. It must be pointed out, however, that the pneumococcus was obtained in pure culture from the spleen at the autopsy. Contrasting with these the four cases of typhoid fever, the polymorphonuclear cells amounted to only 4 per cent. In four examples of bronchopneumonia they averaged 29 per cent. The highest percentage of these cells met with was 40 per cent. Under this heading were included the finely granular cells with transitional nuclei. Phagocytosis of erythrocytes and bacteria by these cells was noted in some instances, but in the majority of cases this function was confined to the endothelial cells. This excess of polymorphonuclear cells occurred in cases which were clinically instances of septic infection. In certain diseases without any such evidence of infection streptococci or staphylococci were cultivated from the splenic juice, but there was no excess of inflammatory leucocytes.

(b) Mononuclear Neutrophiles.—As a general rule, with excess of the finely granular polymorphonuclear cells this variety of cell was present in abnormal numbers. We may quote, for example, a case of acute pneumonia with 33 per cent. of polymorphonuclear and 7.5 per cent. of mononuclear. In a case of epidemic cerebrospinal meningitis there were 9.5 per cent. of the mononuclear neutrophiles and 16 per cent. of the polynuclear. The appearance of these mononuclear cells is precisely similar to that of the small neutrophilic myelocytes of the bone-marrow.

(c) Coarsely Granular Eosinophiles.—These were met with in only small numbers, the highest percentage being only 1.7 per cent., and they resembled those normally found in the blood. An occasional large mononuclear eosinophile was seen.

(d) Lymphocytes. — Small and large lymphocytes were the predominating cells present in the films, their proportion depending on the ratio of other cells present. The only change noted in these cells was the occasional occurrence of nuclear fragmentation.

(e) Endothelial Cells.—An increased number of these cells was met with in most instances of acute bacterial infection. They were of several varieties; in some the nucleus was small and dark and the cytoplasm basophilic; in others the nucleus was larger and paler, the cytoplasm stained less deeply and often contained "azur" granules. In some instances the cells were vacuolated. These cells constitute the main phagocytes of the spleen and were seen ingesting red blood-corpuscles, pigment and bacteria, and at times lymphocytes. Mitosis and amitosis was commonly met with in these cells.

(f) Plasma-cells.—These cells were met with scattered through the sections in most instances and formed a small percentage of the cells present in the films. They were most numerous in a case of chronic endocarditis, where they totalled 4 per cent. They were never seen to be phagocytic.

(g) Giant-cells.—Small giant-cells of the lymphadenoid type were commonly present, but usually in very small numbers. In one case of influenzal pyæmia they were numerous throughout the section. Nothing resembling the large type of giant-cell met with in bone-marrow was ever seen.

(h) Nucleated Red Blood-corpuscles.—In a certain number of instances where anæmia was present during life nucleated red cells, both normoblasts and megaloblasts, were seen in the splenic pulp. Their presence and number could be accounted for by the condition of

the blood during life. In a film preparation from the spleen of one case of pernicious anæmia they were found in large numbers, no fewer than eighty-six normoblasts and forty-six megaloblasts being met with while counting 1,000 white cells.

PHAGOCYTOSIS.

(a) Red Blood-corpuscles.—Previous observers have drawn attention to the frequency with which phagocytosis of red blood-corpuscles occurs in the spleen in certain acute and chronic diseases, especially typhoid and Malta fever. The findings in the present investigation were in accordance with these facts. The cells which take an active part in this phagocytosis are almost entirely endothelial cells which may be seen lying free in the lumina of the sinuses or attached to their walls. This phenomenon was observed in almost every spleen examined, but was much more marked in the case of certain diseases. As the extent of this process varies for the whole organ so it varies markedly as regards individual cells. In some cases the endothelial cells contained only one or two red blood-corpuscles; in others they were distended with red cells. The affinity of the engulfed red cells for acid dyes was often markedly decreased and strongly suggested the effect of hæmolysis. As further evidence of this action, in some instances the splenic sinuses contained, in addition to faintly staining red corpuscles, a granular debris and a homogeneous substance suggesting serum, both of which stained with an acid dye. In Group I, which comprises the acute infective and non-infective diseases, phagocytosis of erythrocytes was often well shown. For instance, in the majority of cases of acute pneumonia (lobal and lobular) a marked degree of phagocytosis was recorded, but in typhoid fever this phenomenon was the outstanding feature of the sections. In the other groups of cases, the best examples of this condition were met with in pernicious anæmia. Here out of a total of eight cases this phenomenon was moderate in one, well marked in three, and present to a striking degree on two occasions.

(b) Pigment.—In the large majority of the cases in which phagocytosis of red cells was well marked the spleen contained large quantities of pigment; this was scattered through the substance of the spleen, inside the wandering endothelial cells or in the cells lining the sinus walls. It was usually present in the form of golden brown or dark brown, occasionally almost black, granules, and gave a striking appearance to the sections. It was readily observed that the degree of pigmentation was

in direct relation to the phagocytosis of the erythrocytes and the apparent hæmolysis. As has previously been observed, the micro-chemical reaction for free iron was of little value in determining the amount of blood-destruction.

(c) Lymphocytes.—In a certain number of cases, mostly examples of acute infections, phagocytosis of lymphocytes by the endothelial cells was noted. In certain other cases, previously referred to, endothelial cells at the centres of the Malpighian bodies were devouring lymphocytes and their remains.



FIG. 3.

Drawing of a portion of the spleen from a case of pernicious anemia; a large amount of pigment, in the form of dark granules, is present chiefly in the cells lining the splenic sinuses. The sinuses are not distended, but the spleen pulp is packed with faintly staining erythrocytes. The spleen was much enlarged, weighing 36 oz. (Zeiss objective, D.D.)

The Histology of the New Bone-formation in a Case of Pulmonary Hypertrophic Osteo-arthritis.

By F. PARKES WEBER.

THE case was described in the Clinical Section of the Royal Society of Medicine on December 11, 1908.¹ The hypertrophic osteo-arthritis was secondary to a mediastinal form of lymphadenoma (Hodgkin's disease), and occurred in a woman, aged 21, who was admitted to the German Hospital in March, 1908, with symmetrical œdema of the arms and legs of some weeks' duration. She had previously enjoyed good health and was not aware of having anything the matter with her beyond the swelling of the limbs. Examination of the thorax, however, showed the presence of a mediastinal tumour, which led to her death in October, 1908. In the hospital the œdema of the extremities gave place to the characteristic appearance of hypertrophic osteo-arthritis, and Röntgen ray examination (Dr. N. S. Finzi) showed this to be of what Dr. H. E. Symes-Thompson² has termed the "diaphysial type" ("osteopathy type"). The joints appeared very little affected. There was no, or very little, accompanying pain, even at a time when the rough projecting surface of the periosteal bony deposit could be easily felt beneath the skin of the forearm. We here reproduce (figs. 1-3) some of the skiagrams kindly made by Dr. Finzi. All the bones of the extremities were affected. In the hands and feet the terminal phalanges and the carpal and tarsal bones appeared least affected. The shadow of the shaft of every long bone was enclosed by a somewhat fainter irregular shadow, evidently due to the formation of new bone under the periosteum. In fact, the diaphysis (in whole or in part) of every long bone was encased by a cylindrical growth of new bone, which in some of the bones of the feet and hands caused the diameter of the shaft to be almost doubled. The distal ends of the femur and humerus were more affected than the proximal, just as has been noted in other cases. Besides all the long bones of the extremities the clavicles were shown by Röntgen rays to have undergone a similar change, but the bones of

¹ *Proc. Roy. Soc. Med.*, 1909, ii, No. 3, Clin. Sect., pp. 66-86.

² H. E. Symes-Thompson, *Med.-Chir. Trans.*, Lond., 1904, lxxxvii., pp. 85-139.

the pelvis, the ribs, and apparently the vertebræ, were not altered; the condition of scapulæ and cranial bones was not examined.

It is, however, the microscopic appearance that I wish specially to refer to. A piece of the upper part of the left humerus was removed for maceration and microscopic examination. The macerated bone



FIG. 1.

Skiagram of the hand showing the increased thickness of the metacarpal and phalangeal bones due to periosteal formation of new bone on the diaphyses.



FIG. 2.

Skiagram of the foot showing the diaphysial formation of new bone in the periosteum, causing great increase in the thickness of the metatarsal and phalangeal bones.



FIG. 3.

Skiagram showing the periosteal new bone-formation on the lower portion of the shaft of the femur.

showed an irregular subperiosteal deposit of new bone on the shaft. Microscopic sections of a decalcified piece were prepared, and Dr. D. Reid at the Lister Institute very kindly made a microphotograph (magnification 15) of one of them (fig. 4). The original compact bone of the shaft, which is relatively darkly stained, is separated by a very open meshwork of bony trabeculae from a less deeply stained outer layer of newly formed bone. The outer surface of this layer of newly formed bone is relatively compact, and appears in section to consist of a series of rather faintly stained bony trabeculae, which in their arrangement are more or less parallel to each other and at right angles to the long axis of the shaft.



FIG. 4.

Microphotograph ($\times 15$) showing the microscopic arrangement of the new bone-formation on the shaft of the humerus. The darkly stained bone in the lower part of the field is the outer surface of the original compact bone of the shaft. The upper part of the field represents the subperiosteal formation of new bone, which is less deeply stained than the older bone.

In most other necropsies on cases of pulmonary hypertrophic osteoarthropathy the bones have, I think, been only macroscopically examined, but the subperiosteal deposit of friable layers of new bone appears to be a feature observed in all cases. Thorburn and Westmacott,¹ after

¹ W. Thorburn and F. H. Westmacott, "The Pathology of Hypertrophic Pulmonary Osteo-Arthropathy," *Trans. Path. Soc. Lond.*, 1896, xlvii, pp. 177-190.

recording the necropsy on their own case and the findings in other published cases, remarked: "We may now safely state that the condition which we have described is one of very extensive periostitis of an extremely chronic type." Moreover, the microscopic structure of the newly formed bone in the present case is apparently similar to that found in cases of chronic periostitis when accompanied by new bone-formation. Yet, clinically, the bone affection, in its absence or relative absence of pain, differs from ordinary kinds of periostitis. Syphilis (at all events, inherited syphilis) may occasionally (as a late manifestation) give rise to a diffuse hypertrophic osteoperiostitis of long bones, with much subperiosteal formation of new bone on the diaphyses, especially of the tibiae,¹ and in such cases the bony change may be almost symmetrical in distribution and relatively painless, but the bones of the hands and feet are not affected in the same way that they are in "pulmonary hypertrophic osteo-arthritis."

¹ Vide F. P. Weber, "A Note on Congenital Syphilitic Osteitis Deformans," *Brit. Journ. Children's Diseases*, Lond., 1906, v, p. 83.

A Note on the Histology of a Case of Myelomatosis (Multiple Myeloma) with Bence-Jones Protein in the Urine (Myelopathic Albumosuria).

By F. PARKES WEBER and J. C. G. LEDINGHAM.

IN a paper on multiple myeloma (myelomatosis) which one of us read before the Royal Medical and Chirurgical Society in 1903¹ records of about forty cases were collected in which the presence of Bence-Jones protein in the urine ("myelopathic albumosuria" of Bradshaw) had been observed. Histological examination of the tumour-like growth in the bone-marrow had already been made in a good number of myeloma cases, and since then several other cases have been examined. In giving histological descriptions, however, it is probable that cases characterized clinically by the presence of myelopathic albumosuria have not been sufficiently distinguished from the other myeloma cases. In some cases, indeed, in which careful histological examinations have been made practically nothing was known of the symptoms during life, especially as to whether there was or was not "myelopathic albumosuria" present at any time. In the case now under consideration there is fortunately no uncertainty in this respect. The condition of the urine was recognized several years ago.

The patient was a woman, aged 65, who was long under the care of Dr. H. Savory in the Bedford County Hospital. Afterwards she was again in the same hospital under the care of Mr. Gifford Nash, and died there on January 25, 1909. Dr. Savory and Dr. Gowland Hopkins, of Cambridge, carried out most careful investigations with regard to the influence of diet on the protein excretion, and we doubt whether the metabolism in any other case of this disease has ever been studied so thoroughly. One of us (F. Parkes Weber) was kindly allowed to make the post-mortem examination at Bedford in conjunction with Dr. Gowland Hopkins, and it is by the courtesy of Dr. Savory and Dr. Hopkins that we have been permitted to publish this note on the histological features. They will shortly publish a detailed account of the case, and in regard to the clinical features it is sufficient to add that, as they tell us, the protein excreted in the urine was of typical character and averaged 12 grm. per diem.

¹ F. P. Weber, *Med.-Chir. Trans.*, Lond., 1903, lxxxvi, pp. 395-467.

NECROPSY.

The body was that of a medium-sized, rather emaciated female with considerable kyphosis of the lower dorsal region and apparently abnormal projection of the right great trochanter. The head and skull were not examined. We shall for convenience first describe the macroscopic changes noted in the bones and bone-marrow and then proceed to the viscera and the histological examination of the growth.

The bones examined included the sternum, ribs, clavicles and part of the vertebral column, part of the right humerus and part of the right femur. The sternum, ribs, clavicles, humerus and femur were all found transformed into mere shells of very hard, brittle, compact bone filled with a dark red opaque jelly-like substance which replaced the normal bone-marrow. Not only were the medullary canals of the long bones greatly enlarged at the expense of their osseous walls, but much of the cancellous tissue of the bones (*e.g.*, of the head of the humerus) was found to have been replaced by the dark red opaque jelly-like substance in question. If it is permissible to speak of this altered bone-marrow as bone-marrow at all, evidently the total amount of bone-marrow in the body must have been immensely in excess of the normal amount. There were no local bulgings on the ribs or sternum, nor were any fractures detected such as have been noted at necropsies in some cases, but, on the contrary, it was seen that a former fracture of the surgical neck of the right humerus (which had occurred a few months before the patient's death) was completely healed by good bony union of the fragments. The projection of the right great trochanter was found to be due to the fact that the neck of the femur, though shortened, was nearly at right angles to the shaft, as if it were the site of an old fracture. The portion of the vertebral column examined was the lower dorsal region—that is to say, the site of the greatest kyphotic bend. The bodies of the vertebræ consisted of hard bony shells containing only a remnant of the normal cancellous tissue, much of the cancellous bone having been replaced by a red opaque jelly-like substance exactly similar to that already found in the other bones. This jelly-like substance, or altered bone-marrow, was seen on microscopic examination to consist of a diffuse tumour-like growth, with fat-cells and normal bone-marrow constituents interspersed amongst the collections of the cells proper to the growth. Histologically the growth was a "plasmacytoma," "plasmoma," or "myeloma plasmacellulare," but we shall return to the histological

features of the growth after describing the results of macroscopic and microscopic examination of the viscera.

The heart (weight $9\frac{1}{2}$ oz.) showed nothing abnormal. The aorta, as much of it as was examined, showed no excessive atheroma, excepting some calcification quite at its commencement, close to the aortic valves. There were a few old pleuritic adhesions. There was hypostatic congestion of the bases of the lungs, and the hardened condition of the left lower lobe suggested the presence of actual pneumonia. Microscopic examination of this part of the lung proved that there was pneumonia and that the pulmonary alveoli were

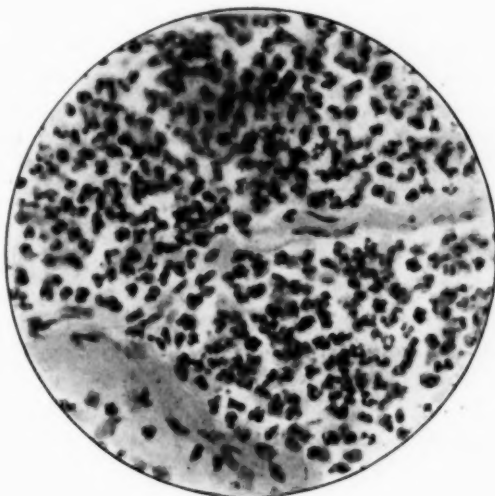


FIG. 1.

Microphotograph showing the pneumonia. (Magnification, 500.)

typically infiltrated with pus-cells (fig. 1). Examination of blood-films from the blood in the heart gave the following differential count (J. C. G. Ledingham) of white cells: small lymphocytes 1·2 per cent.; large lymphocytes 19 per cent.; large mononuclears and transitionals 0·6 per cent.; polymorphonuclears 77·3 per cent.; eosinophiles 0; myelocytes 1·8 per cent. A good many of the polymorphonuclears appeared immature, approaching the myelocyte type. No plasma-cells could be distinguished. No nucleated red cells were seen. No changes were observed in the red cells.

Macroscopic and microscopic examination of the liver (weight 51 oz.) and pancreas showed no disease. There was no cholelithiasis. The spleen (weight 3 oz.) was small and seemed rather shrivelled. On cutting into it there appeared to be an increase of the fibrous trabeculae; microscopic examination showed a certain amount of fibrosis and thickening of the walls of the arteries. The suprarenal glands were not examined.

The kidneys weighed together 12 oz. One was slightly bigger than the other, but there was no macroscopic evidence of disease. Microscopic examination showed nothing abnormal beyond scattered spots of a chronic interstitial fibrotic change of very slight degree. The urinary bladder appeared healthy. The body of the uterus contained a mass of fibroids, several of them calcified, and the uterine canal was elongated to about 5 in. The ovaries were shrivelled, and the right one contained a cherry-sized cyst. There was a right parovarian cyst filled with clear straw-coloured fluid. The stomach and intestines, as far as they were examined (small intestine examined microscopically), appeared normal. Ordinary hæmatoxylin sections of the lowest dorsal segments of the spinal cord were pronounced by Dr. Gordon Holmes, who kindly examined them for us, to show nothing abnormal, except swelling of the dorsal root-fibres in the root-entry zone, suggesting slight compression of the dorsal roots; in one section there was evidence of slight compression of one lateral surface of the cord.

There was no evidence of metastasis of the bone-marrow growth anywhere in the body outside the bones. No enlargement of any lymphatic glands was observed.

EXAMINATION OF THE BONE-MARROW TUMOUR-LIKE GROWTH (J. C. G. LEDINGHAM).

Pieces of the marrow from the femur, humerus and sternum were fixed in Orth's fluid and embedded in paraffin. The stains employed were Laurent's methylene-blue-eosin mixture, Unna-Pappenheim's methyl-green-pyronin, Twort's Licht-Grün-neutral-red mixture and hæmatoxylin-orange-rubin.

The microscopical features of the marrow were essentially the same in all three situations, but the following description will apply mainly to the humerus marrow, in which the peculiar cell-infiltrates constituting pathological change occurred in their most characteristic form. Examination of the sections even with the naked eye shows irregular densely

stained areas standing out prominently among a looser and less cellular tissue. Microscopically (figs. 2-4) the fat-cells of the marrow are still intact, and in some places appear to have elbowed out the other cellular elements. Fat-spaces are less numerous, however, in the densely stained areas; these present a somewhat tessellated appearance, consisting of cells of uniform type arranged in columns or rows around the still remaining fat-spaces; frequently between two adjacent fat-spaces the intervening infiltrating cells appear to be laterally compressed and of smaller dimensions. Otherwise these cells, though conforming to the same type, show extraordinary variations in size, nuclear arrangement, and condition of cytoplasm.

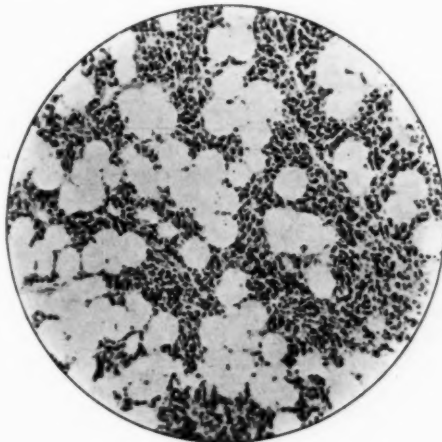


FIG. 2.

Microphotograph showing the bone-marrow growth. (Magnification, 100.)

With the Unna-Pappenheim stain (Plate I) the cells present all the morphological characters of plasma-cells. The nucleus is wheel-shaped, with the chromatin granules arranged mostly at the periphery. In the centre of the nucleus is usually seen a clear space which lodges the nucleolus, a round, sharply defined body, which stains a bright red in contrast to the green nuclear chromatin. The cytoplasm is often voluminous, with intensely basophil reaction. In many cases the cytoplasm is highly vacuolated, the intervening granular substance staining deeply with the pyronin. Round the nucleus, which, as a rule, is placed eccentrically, is the clear unstained "halo" characteristic of plasma-cells.

The nucleus may be single or double, and some large forms have been noted with three nuclei. Mitotic figures are only rarely met with in these cells, and when present do not conform to the usual type. In Plate III are drawings of three cells with mitotic figures. It will be noticed that during mitosis the basophilia of the cytoplasm is greatly reduced and the chromatin is arranged in the centre of the cell as a twisted highly pyknotic band.

Some of the cells (Plate II) show a peculiar partition of the nuclear chromatin into three or four spindle-shaped, densely stained masses, which may represent an early phase in the mitosis. In the case

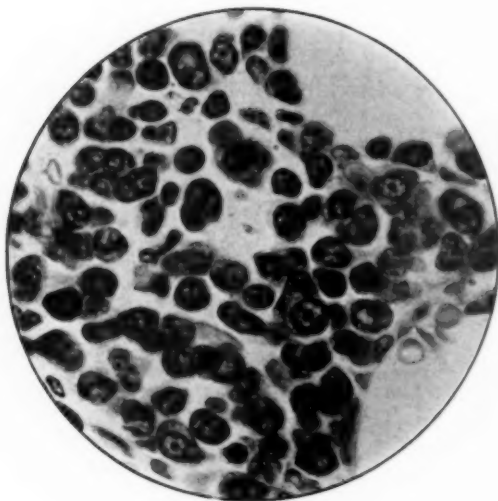


FIG. 3.

Microphotograph showing the bone-marrow growth. (Magnification, 750.)

reported by J. H. Wright (*Johns Hopkins Hospital Reports*, 1900, ix, p. 359¹) no mitotic forms were seen, but a few of the cells contained elongated, hour-glass or dumb-bell nuclei. These evidently correspond to the mitotic forms in our case. Wright also noted cells with two

¹ In Rudolf Hoffmann's case, as well as in Wright's, the myelomatous growth was found to be histologically a "plasmacytoma," or "plasmoma," as in the present case. *Vide Hoffmann, "Ueber das Myelom, mit besonderer Berücksichtigung des malignen Plasmoms," Beiträge zur path. Anat. und. zur allg. Path., Jena, 1903, xxxv., p. 317.* Since then other examples of "myeloma plasmacellulare" have been described (see Addendum).

nuclei connected by a thin band of chromatin, suggesting amitosis (direct division; that is to say, division other than by karyokinesis). Evidence of direct division was not found in our case.

It is remarkable that so few mitotic forms occur. As a matter of fact, degeneration of these plasma-cells is more in evidence than active proliferation. The nucleus in numerous cases is seen to be undergoing lysis (Plate I), and frequently one or two spots of chromatin are all that remain of the nucleus. The cytoplasmic residue still retains, however, its marked affinity for pyronin. Lying alongside perfect cells are seen numerous cytoplasmic cell-residues staining deeply with pyronin and containing no trace of a nucleus.

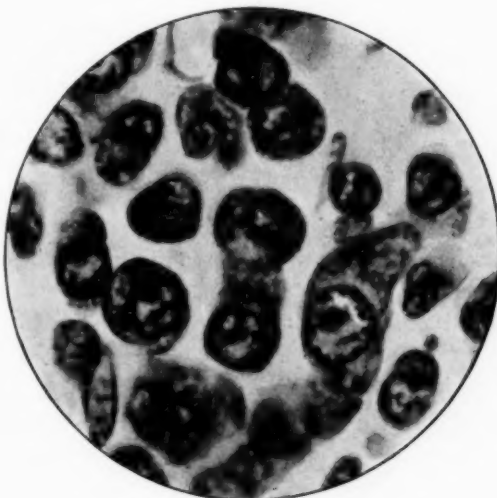


FIG. 4.

Microphotograph showing the bone-marrow growth. (Magnification, 1,500.)

With Twort's stain the various elements of the plasma-cell are thus differentiated. The nuclear chromatin and the granular portion of the cytoplasm stain a brick-red. The nucleolus is stained a light red. The perinuclear "halo," the intranuclear space, and the substance contained in the cytoplasmic vacuoles take up the green or acid stain (Plate III). This green staining of the nucleoplasm is an interesting feature. It has been noted by one of us (J. C. G. Ledingham) in the nucleus of the *Entamoeba histolytica*. The perinuclear "halo" of the

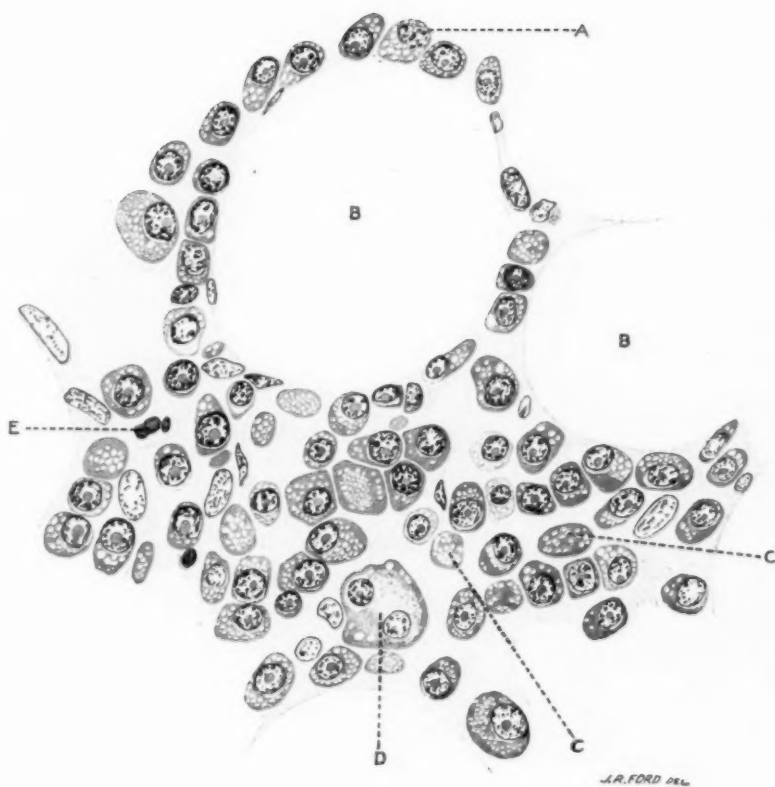
EXPLANATION OF PLATE I.

SECTION OF PART OF THE BONE-MARROW GROWTH.

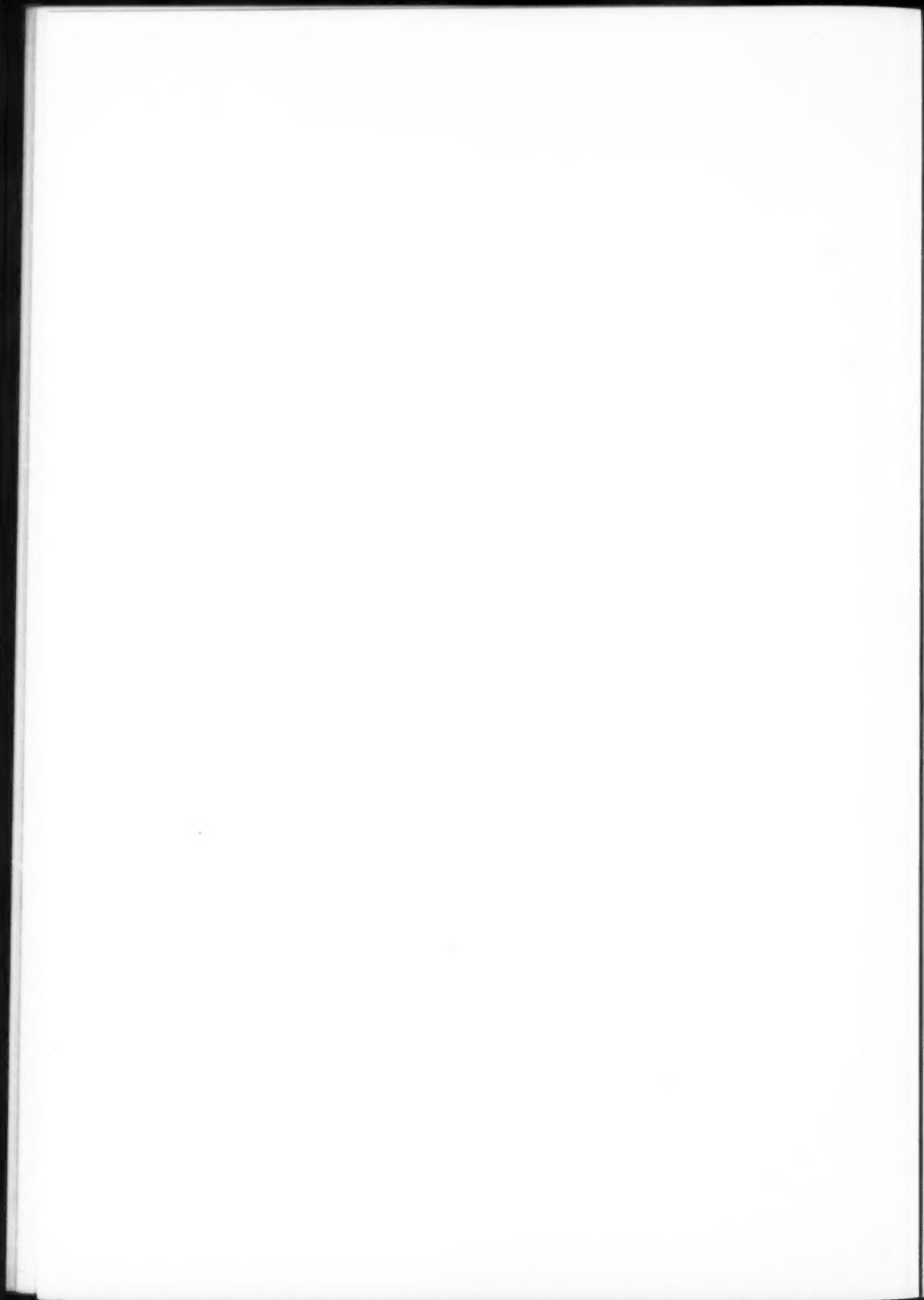
(Magnification, 650.)

Methyl-green-pyronin stain (Unna-Pappenheim method). The plasmacyte infiltrate surrounds the fat-vesicles. The vacuolated cytoplasm and the nucleoli (stained red) of the plasma-cells are well shown.

- A—Plasma-cell in process of karyolysis.
- B—Fat-vesicle.
- C—Cytoplasmic remnant of plasma-cell after complete karyolysis.
- D—Large kind of plasma-cell with double nucleus and well-marked perinuclear "halo."
- E—Erythroblast with lobed nucleus.



PARKES WEBER & LEDINGHAM: *Histology of a Case of Myelomatosis.* Plate I.



plasma-cells remains after complete karyolysis as a green irregular space (Plate III). With Laurent's stain (Plate II) the perinuclear "halo" is very well marked, but remains unstained. The nucleolus is stained a bright pink in contrast to the dark blue nuclear chromatin.

Outside the dense infiltrates all varieties of the ordinary marrow elements are present, together with plasma-cells, which find their way into all parts of the marrow. The adventitial sheaths of the vessels are invariably surrounded by rows of plasma-cells. Eosinophil myelocytes are very numerous. Small erythroblastic foci are seen here and there. The giant-cells of the marrow are not increased in number. No micro-organisms of any kind were detected, and staining by Schmorl's method revealed no spirochaetes.

The question now arises, Does the histological evidence afford any clue to the source of the Bence-Jones albumosuria? In Weber's case (1903), which was investigated chemically by Hutchison and MacLeod, no body giving exactly the same reactions as those of Bence-Jones protein was found in the bones, blood, or organs. From the vertebrae and ends of the femur, however, a protein was obtained giving very similar reactions, differing somewhat in the temperature at which it coagulates, and in not being redissolved on boiling. Moreover, no protein like that detected in the myelomatous marrow could be isolated with the same methods from normal marrow.

CONCLUSION.

From the histological evidence in the present case we are inclined to offer the suggestion that the cytoplasmic residua of karyolyzed plasma-cells may be the source of this peculiar protein. It must be remembered that the total quantity of the (myelomatous) bone-marrow is, as we have already pointed out, greatly in excess of the normal. Moreover, the position (in the bone-marrow) of the tumour-like growth shows that metabolic or degenerative products of the plasma-cells, of which it consists, must readily gain access to the circulating blood-stream; and from the blood it has been shown experimentally that Bence-Jones protein is readily (like haemoglobin or glucose, when present) excreted through the renal filter with the urine, or at least, according to von Decastello, if the renal tissue is in any way damaged.¹ In regard to the relationship of myelopathic albumosuria to the bone-marrow disease, we will merely

¹ On this point see A. von Decastello, *Verhandl. d. Kongresses für inn. Med.*, Wiesb., 1908, xxv., p. 620.

EXPLANATION OF PLATE II.

SECTION OF PART OF THE BONE-MARROW GROWTH.

(Magnification, 900.)

Laurent's eosin-methylene-blue mixture stain. Shows plasma-cells of the growth, especially very large forms of the plasma-cells, between fat-vesicles.

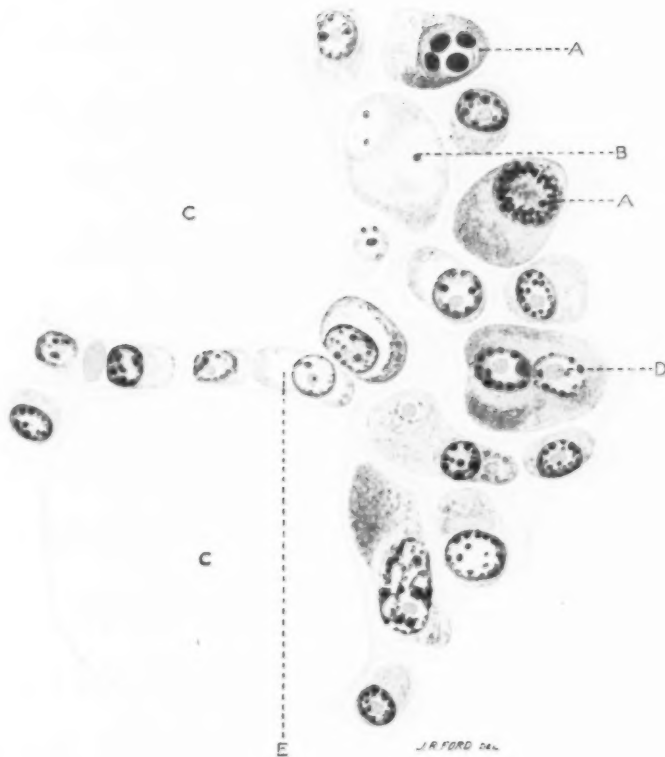
A—Form of plasma-cell, showing peculiar position of the nuclear chromatin (? prophase of karyokinesis).

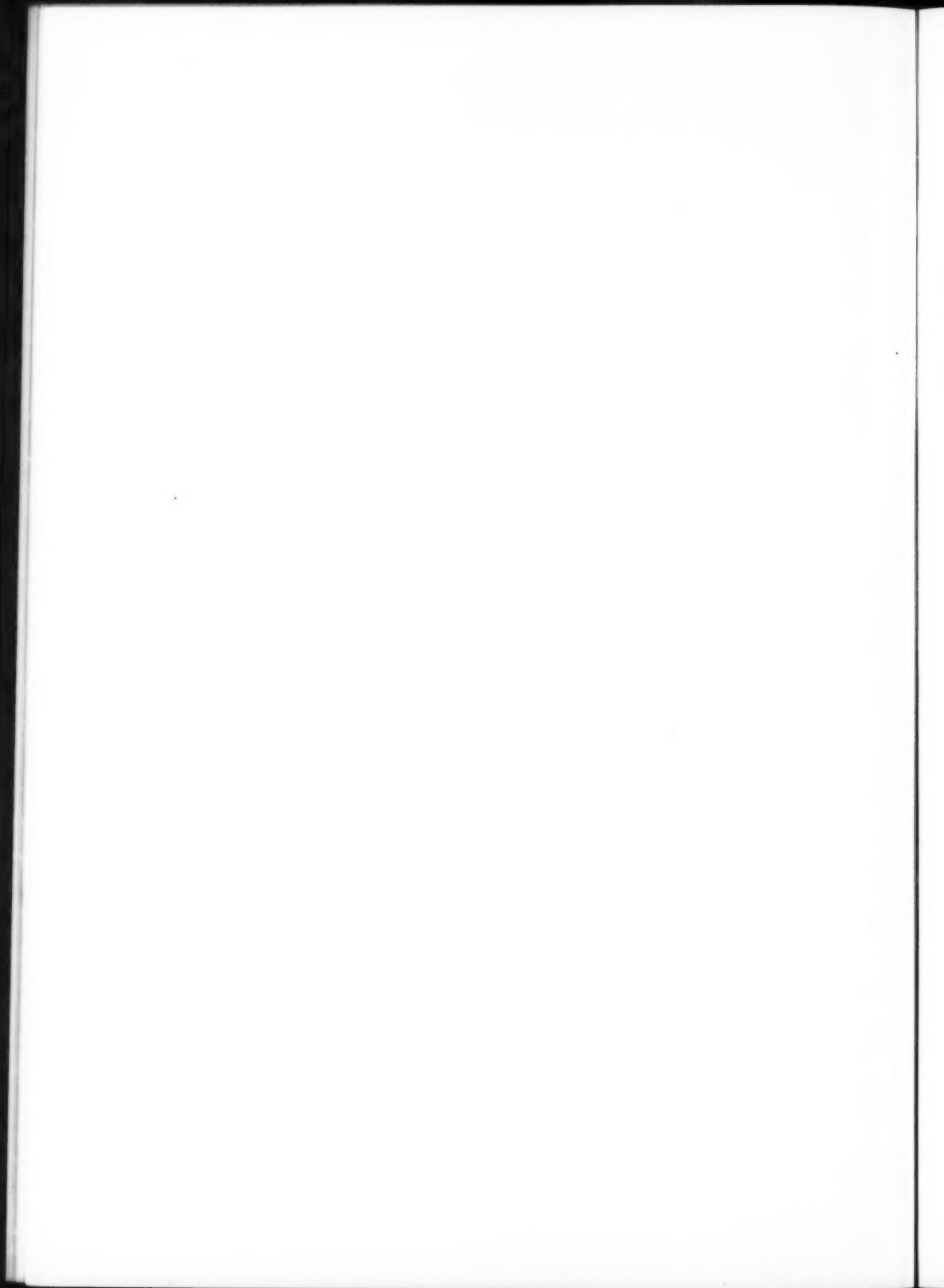
B—Plasma-cell, showing extreme karyolysis.

C—Fat-vesicle.

D—Nucleolus, stained pink, lying in clear intranuclear (perinucleolar) space.

E—Perinuclear "halo."





remark that, for practical purposes, whenever a copious and persistent excretion of Bence-Jones protein in the urine has been observed, the patient has been sooner or later found to be affected with diffuse primary tumour-formation of the bone-marrow. Only one or two apparently genuine exceptions to this rule have as yet been recorded.¹

One of us (F. Parkes Weber) in the paper of 1903 (already alluded to) ventured to draw an analogy between the bone disease, "myelomatosis," on the one hand, and the skin disease, "mycosis fungoides," on the other. The justification of such an analogy becomes evident now that it is known that in at least some cases of myeloma the growth consists of plasma-cells, whilst in mycosis fungoides the sarcoma-like tumours of the skin have been found to be "plasmomata."

ADDENDUM—CONSIDERATION OF THE TERM MYELOMA.

There is no occasion to take up unnecessary space here by referring individually to all the recorded cases of myeloma. A review of all those described previously to 1903 will be found in the papers already referred to by F. P. Weber and R. Hoffmann. The histology of more recent cases has been recorded or discussed by MacCallum, Lubarsch, Jellinek, Menne, Abrikossoff, Saltykow, Sternberg, Permin, Aschoff, Scheele and Herzheimer, Ribbert, Simmonds, v. Verebely, Hueter, Charles and Sanguinetti, degli Occhi, H. A. Christian, Umber, Tschistovitch and Kolesnikowa, A. Herz, and Benda (*see* references at the end of the paper).

The main points round which discussion has centred have been the differentiation of the various forms of bone-marrow growth, the presence or absence of metastases, and the morphology of the cells constituting the bone-marrow growth. A myeloma has been defined as a primary diffuse or multiple tumour of the bone-marrow, consisting of elements peculiar to that tissue and giving rise to no metastatic growths. Cases which do not conform to this description, either because of peculiar histological structure or because of the occurrence of metastases, would be excluded from the group. These criteria were employed by Menne in his arrangement of the cases recorded up to 1906.

It might certainly be advisable at present to exclude those rare cases of alveolar sarcoma and endothelioma which have been recorded, but it does not seem possible to exclude, on the ground of the occurrence of metastases, those cases in which the tumour elements are at any rate

¹ See von Decastello, *loc. cit.*

EXPLANATION OF PLATE III.

SOME ISOLATED CELLS OF THE BONE-MARROW GROWTH.

(Magnification, 900.)

In the two upper figures Twort's Licht-Grün-neutral-red mixture has been used, and marked differential staining has been obtained.

A—Cytoplasm, stained light brown, studded with vacuoles, the contents of some of which have taken on the green (acid) dye.

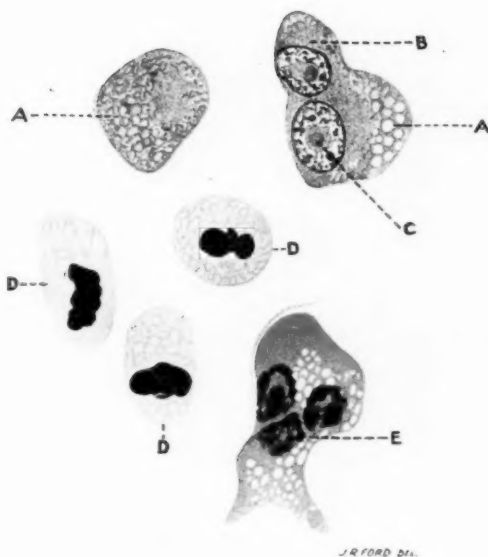
B—Perinuclear "halo," also stained green.

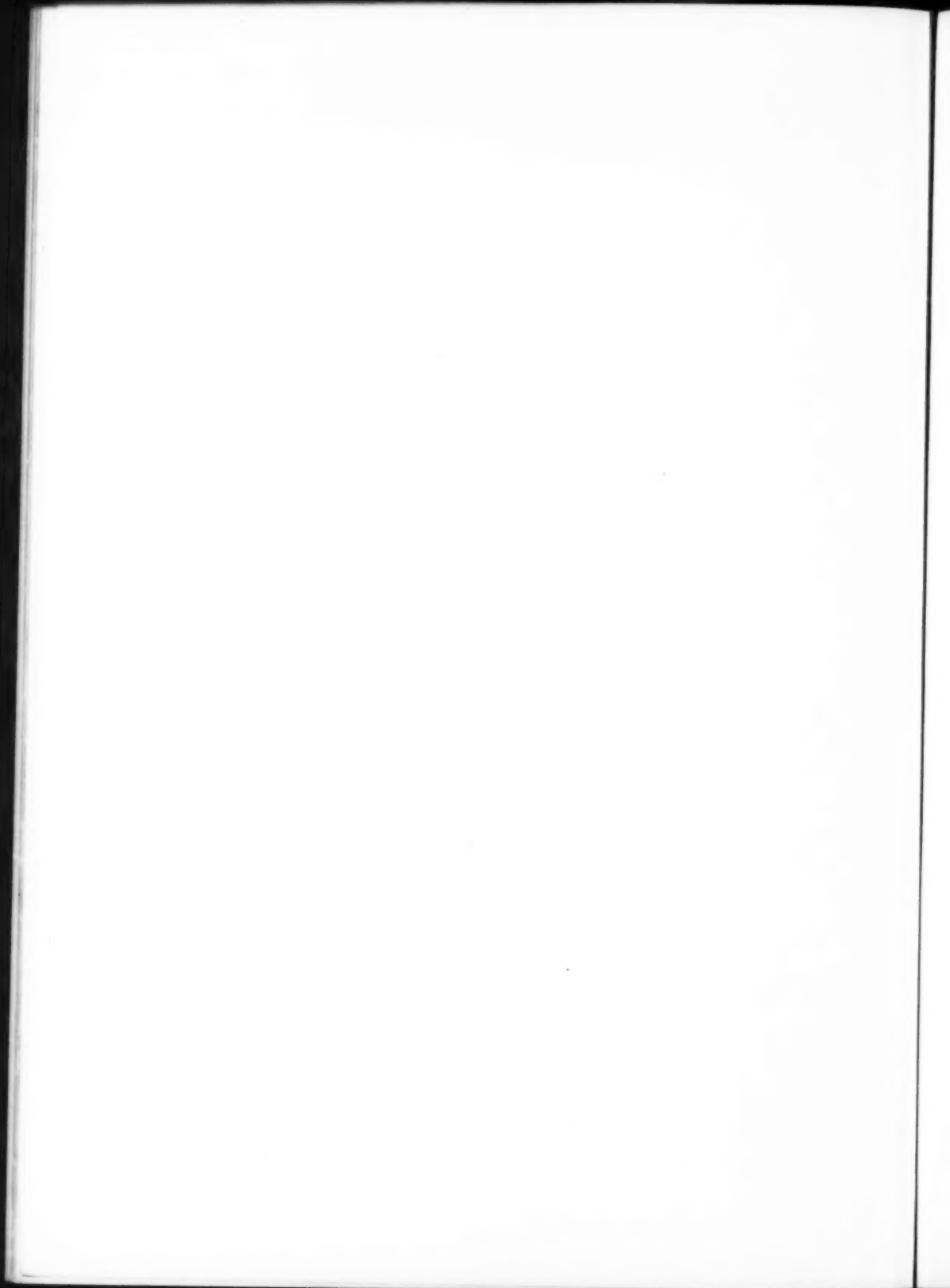
C—Nucleolus, stained light brown, surrounded by the intranuclear (perinucleolar) space, the contents of which have likewise taken up the green stain. The nuclear chromatin is stained reddish-brown. In the upper left figure the nucleus has undergone complete karyolysis; but the perinuclear "halo" (green) remains.

The four lower cells have been stained with methyl-green-pyronin (Unna-Pappenheim).

D—Peculiar mitotic form with twisted pyknotic nucleus. The cytoplasm (coloured pink) shows a reduced affinity for the pyronin (that is to say, diminished basophilia) during the process of mitosis.

E—Large unusual form of tumour-cell with triple nucleus undergoing pyknosis.





not foreign to the normal bone-marrow. If one regards the disease as a systemic affection of the hæmatopoietic apparatus the objection to the inclusion of those very few cases associated with metastases falls to the ground. With our modern views as to the occurrence of bone-marrow elements in organs like the spleen and liver under certain conditions (myeloid transformation), the occasional presence in these organs of so-called metastatic nodules can readily be explained.

It would seem to us of far more value to classify the cases of myeloma according to the presence or absence of Bence-Jones protein in the urine, but we cannot discuss this question here. A very convenient, but probably artificial, classification can be made on the basis of the morphology of the tumour-cells. Five types would thus be distinguished, viz:—

- (1) *Lymphocytic* (Benda's case ?).¹
- (2) *Pre-myelocytic*, or *Myeloblastic*.—The cells in these cases lack the definite granulation of the myelocytes, but in most respects resemble the non-granular "pre-myelocytes" or "myeloblasts." To this type apparently belong the cases of Tschistovitsch and Kolesnikowa, Hueter, MacCallum (1905), Abrikossoff, Saltykow, Menne, and Permin.
- (3) *Myelocytic*.—To this type several cases have been found to conform, but even in these the granulation of the myelocyte-like tumour-cells is generally not completely developed. Besides F. P. Weber's case (1903) the cases of Sternberg (1903), Charles and Sanguinetti (1907), A. Herz (1908) and Umber (1908) apparently belong to this group.
- (4) *Erythroblastic*.—Ribbert's case of this supposed character remains isolated.
- (5) *Plasmacytic*.—Since the cases (already referred to) of Wright (1900) and Hoffmann (1903), several myeloma cases have been described in which the growth has been found to consist of plasma-cells or of cells closely resembling plasma-cells (Aschoff, v. Verebely, H. A. Christian, degli Occhi).

The plasma-cell growth has been variously described as malignant "plasmoma," "plasmacytoma" or "myeloma plasmacellulare." Our present case constitutes about the seventh belonging to this category. Unlike our case and that of Wright, Hoffmann's case presented a so-called metastatic nodule in the liver consisting of plasma-cells.

¹ In F. P. Weber's case of 1896 (*Trans. Path. Soc. Lond.*, 1897, xlviii, p. 169) no special staining for plasma-cells was undertaken till 1903. Then, however, Dr. J. M. H. MacLeod found that the cells of the bone-marrow growth showed a greater resemblance to lymphocytes than to the typical plasma-cells of the granulomata.

Wright observed that plasma-cells occur in the normal bone-marrow, and we now know that they may occur in all organs. Myeloid transformations may sometimes, as already stated, occur in the spleen, lymphatic glands and liver. Hence, as Lubarsch remarks, the occurrence of "metastatic" nodules consisting of myelocyte-like cells or plasma-cells would readily fit in with the conception of myeloma as a systemic affection.

At present, however, the time is not ripe for the adoption of very dogmatic views as to the exact place of myeloma in the classification of diffuse affections involving the hæmatopoietic apparatus.

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Pathological Section.

March 16, 1909.

Mr. S. G. SHATTOCK, President of the Section, in the Chair.

On Normal Tumour-like Formations of Fat in Man and the Lower Animals.

By S. G. SHATTOCK.

WHETHER there is really a difference in the physiological behaviour of the fat of a lipoma and that of the rest of the body is a question for the answer to which more evidence is needed than is at present forthcoming. The belief rests mainly upon a specimen in the museum of St. George's Hospital, where there were lipomata in the mesentery of a phthisical patient from whom nearly all the natural fat had been removed; and upon a case (of Schuh) where huge masses of fat existed on the head, throat, and chest of a man whose abdomen and legs were extremely thin.

From these data Paget¹ has deduced an independence for the life-history of lipomata as contrasted with the rest of the fat. The observations, were they beyond dispute, would illustrate the anarchy of a new growth so simple indeed as a lipoma, which not only increases without reference to the general requirements of the body, but would withstand the calls made upon it as reserve material in time of need. Dr. Parkes Weber² has recorded a case of lipomatosis of the neck, arms, and pubic region in a man with slight general wasting. The patient was a publican. Here the accumulation of fat must be associated, presumably, with chronic alcoholism, and the wasting, as Dr. Weber observes, was probably due to hepatic cirrhosis. Schuh's case may have been of the same class.

¹ "Lectures on Surgical Pathology," 3rd ed., 1870, p. 378.

² *Trans. Clinical Soc., Lond.*, 1904, xxxvii, p. 220.

Through the kindness of Dr. R. S. Trevor I have been able to examine the preparation in St. George's Hospital Museum. The specimen (Series XVII, No. 21) shows three oval swellings in a portion of mesentery, and a more considerable isolated tumour suspended above it. The isolated growth is oval in shape, 9 cm. in its longer diameter, and obviously consists of fat. Of the three in the mesentery, one is 2.5 cm. in its longer diameter, one is 2 cm., and the third is 1.5 cm. That last referred to has a suspicious want of transparency, and on being divided was found to be firm, with irregular caseous foci suggestive of a tuberculous mesenteric gland, a suggestion confirmed by microscopic examination which shows the presence of large numbers of confluent giant-cell systems undergoing caseation. The swelling 2.5 cm. long, on being cut through, was obviously of transparent, soft, pale yellow fat; and that 2 cm. in length was clearly of the same character. In the older edition of the catalogue the tumours are erroneously stated to be in the omentum. The largest may have come from the omentum; the others are in the mesentery. The patient was a woman, aged 28, under the care of Mr. Hawkins in 1843. She was greatly emaciated, and a swelling could be felt through the parietes on the right side of the abdomen. After death the lungs were found studded with tubercles and small cavities, most numerous in the upper portions. The right kidney was tuberculous; the upper end of the ureter, thickened; the interior of the bladder was ulcerated.

Intra-abdominal accumulations of fat of a localized kind are not uncommon amongst Ophidia and Reptilia, where they serve as food reserves. In the human subject the subperitoneal fat around the kidney is the last to be removed; and it is difficult to believe that the fat in the tumours just referred to would permanently have withstood absorption. But to cite something more positive. A short while ago a gentleman consulted Dr. H. P. Hawkins for the reason that he was steadily becoming thinner. And he stated, as a curious thing, that during this time a fatty tumour which had existed for some years on the left shoulder had almost disappeared. The tumour was just palpable on the left side at the junction of the neck and the shoulder. The condition from which the patient was suffering was profuse expectoration, not tubercular, and probably bronchiectatic. The reduction of a lipoma during emaciation has its parallel in that of a uterine fibromyoma when the uterus itself undergoes senile atrophy.

The tumour-like formations of fat which form the subject of the present communication are certainly normal in their physiological

behaviour, for they are utilized whenever the general needs of the body require it. Yet without the knowledge that such enlargements are "natural" to certain groups of animals, and not rare or isolated conditions, they would certainly be classed anatomically amongst lipomata; and, what is more, they are not all "diffuse" in kind, for some are as well defined as the most "circumscribed" of fatty tumours.

Steatopygy.

The prominent accumulations of fat on the buttocks of the Bushman and allied Hottentot may be cited first as being the most notable human example of such local obesity. The Bushmen, a short, yellow-skinned people, living chiefly by the chase, are now regarded as the primitive inhabitants of South Africa. But at a remote period their territory appears to have been much more extensive, and to have comprised the whole of the Eastern side of the continent. By the pressure of Bantu races from the north this territory has been curtailed, and the Bushman race displaced to its present position in the South and South-West. The Bantus, at present occupying the whole of the southern third of the continent with the exception of the Hottentot-Bushman region, are West African Negroes crossed with the Hamitic variety of the Caucasian; and by intermarriage of Bantu and Bushman there has resulted the Hottentot, who differs from the Bushman in the black colour of his skin. In the rude native paintings made by South African Bushmen on the rock walls of the caves in which they dwell, the gluteal hump is prominently depicted, the thigh itself being shown slender. Copies of such drawings may be seen in the ethnological collection of the British Museum, Bloomsbury. The copies by L. Tylow, 1893, of similar native rock paintings from the Bushman's River, Natal, in the Pitt-Rivers Museum, Oxford, show the same localization of the eminence, unassociated with any general enlargement of the thigh. The nature of these eminences was verified by Cuvier from the examination of a Bushwoman who died in Paris, where she had been exhibited as the Hottentot Venus.¹ When she was being painted from the nude it could be made out that the protuberance was not muscular, but an elastic, trembling mass beneath the skin, which vibrated with all her movements. The cadaver was thoroughly examined. Cuvier adds that the

¹ Cuvier: "Mémoires du Muséum," iii, 1817. "Extrait d'Observations faites sur le cadavre d'une femme connue à Paris et à Londres sous le nom de Vénus Hottentotte."

eminence consisted only of fat traversed by strong cellular fibres, and that it could be easily raised from the great glutei muscles. Two excellent figures of this woman are given in the "*Histoire Naturelle des Mammifères*," by Geoffroy Saint-Hilaire and Frédéric Cuvier, 1824, the particular article in this case being furnished by Baron (G.) Cuvier himself (Tome I.: "*Femme de race Boschismanne*"). The colour of the skin is brownish yellow, the individual being a Bushwoman, and not, properly speaking, a Hottentot. Viewed from the front, the hips are very broad from the lateral extension of the gluteal masses, but the enlargement does not reach below the upper half of the thigh. The profile view shows that the eminence is properly gluteal; its lower border slightly overlaps the thigh below. The thigh itself is not notably enlarged, *i.e.*, the gluteal deposition is not merely part of a general obesity of the limb, but a proper superadded elevation. This woman whilst living at the Cape, where she was taken under protection at the age of 10, was well fed. She was 26 years of age when exhibited in Paris, and lived there for eighteen months; death occurred from some inflammatory and eruptive disease. It is of importance to bear in mind the type of steatopygy illustrated in these examples, in connexion with other forms of enlargement to be presently referred to.

This condition of steatopygy or steatopygia, as it has been termed, although now most common in the South and South-west, is not rigidly confined to this particular area of the African continent. Not only does a widely diffused Hottentot-Bushman geographical terminology attest the former range of this primitive race over the whole of South Africa as far north as the Zambesi River, writes Sir Harry Johnston, but traces of Bushmen were discovered by this traveller as far north as Lakes Nyassa and Tanganyika. It would seem, he remarks,¹ as if the earliest known race inhabiting what is now British Central Africa was akin to the Bushman-Hottentot type of Negro. Sir Harry Johnston informs me that he has seen a few examples of steatopygy amongst the Pygmy women in North-east Congoland, but not of a very pronounced degree. This race is not now regarded by anthropologists as related to the Bushman. The same authority adds that he has seen a few instances, moreover, of the condition in the Sudanese Negresses of the Mountain Nile, but not reaching the grade observed in the Hottentot-Bushman women. On the whole the character is more confined to the Eastern

¹ "*British Central Africa*," 1897.

than to the Western half of Africa, states the author cited, who informs me also that he is inclined to dissociate the Negro race in general from steatopygy of the Hottentot form. Protruding buttocks, he writes, are much admired by African men as a feminine feature; women, however, do not admire them in a man, but consider him effeminate. The same author informs me that there is a considerable growth of the proper

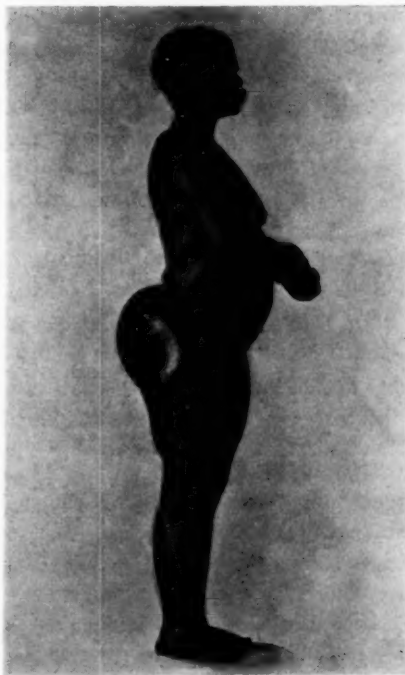


FIG. 1.

A photograph from a cast of a Hottentot woman in the museum of the Royal College of Surgeons. The cast of the male, in the same museum, shows an equally pronounced gluteal eminence. The casts were made from life (in London) by Brucciani in 1851.

steatopygous eminence before and after puberty in the female; that he has never seen it in children, and that the protuberance becomes flabby as a result of deprivation.

In the Central African Nilotic Bongo people, studied by Schweinfurth,¹ this writer states: "All full-grown women attain such an astonishing growth of body, and acquire such a cumbrous superabundance of flesh, that it is impossible to look at them without observing their disproportion to the men. The thighs are very often as large as a man's chest, and their measurement across the hips can hardly fail to recall the picture in Cuvier's atlas of the now famous 'Hottentot Venus.' Shapes developed to this magnitude are no longer the exclusive privilege of the Hottentots; day after day I saw them among the Bongo, and they may well demand to be technically described as 'Steatopyga.' In certain attitudes—as, for instance, when they are carrying their heavy water-jars upon their head—they seem to assume the shape of an inverted S." Schweinfurth gives the figure of a Bongo woman, from behind (vol. ii, p. 121), with a great roll of fat round the abdomen and on the buttocks; the rest of the thigh and the leg below the knee are of no unusual size, nor are the shoulders or upper limbs.

These localized accumulations of fat must not be confused with the general obesity of the women produced in certain African districts by overfeeding. Thus Speke,² in the record of his observations, more eastwards in the Lake Nyanza district, refers to the "wife-fattening" practised at the Court in Karague. He went to visit the eldest brother of King Rumanika, whom he found sitting side by side with his chief wife, with numerous wooden pots of milk; she could not rise, and so large were her arms that between the joints the flesh hung down like large loose-stuffed puddings. On his asking the meaning of the milk-pots, he was told that this was all the product of those pots, as it was the fashion at court to have very fat wives. The dimensions given by Speke of one of the sisters-in-law of Rumanika are: Round the arm, 1 ft. 11 in.; chest, 4 ft. 4 in.; thigh, 2 ft. 7 in.; calf, 1 ft. 8 in.; height, 5 ft. 8 in. These are ordinary examples of general obesity.

But besides the proper gluteal eminence or true steatopygy, Sir Harry Johnston³ states that in the Forest Negroes (one of the groups composing, with Negrito, Bushmen (?), and Nilotic Negroes, the mixed Congo people) there may be an excessive breadth developed transversely across the pelvis and thighs, without any special prominence of the

¹ "The Heart of Africa," Georg Schweinfurth, 1868 to 1871, i, p. 295, English translation of 1873.

² "Journal of Discovery of the Source of the Nile," 1864, chap. viii.

³ "George Grenfel and the Congo," by Sir Harry Johnston, 1908, ii, p. 511.

buttocks. "In almost every physical feature the Forest Negro is the opposite pole to the Bushman in the sphere of the Negro species."

ANCIENT "STEATOPYGOUS" FIGURES: EGYPTIAN.

Amongst the wealth of interest attaching to Professor Flinders Petrie's explorations in Egypt is the discovery of "steatopygous" figures of clay and of mud in certain of the predynastic graves at the villages of Naqada and Ballas, on the western side of the Nile, a short way north of Thebes. Some such are contained in the collection at University College, London; others are in the Ashmolean Museum, Oxford. The fact that all the figures are female is taken to indicate that the representations are those of a conquered race, the captured males of which were killed. The invading predynastic race, according to Professor Petrie's well-known view, was of Libyan source. A few quotations will indicate the chief observations as yet extant, bearing upon this subject.¹ In one tomb of the regular type there were found two female figures of clay, together with some pottery. "The arms are not represented on these figures, and the upper part of the body is slight in comparison with the very bulky thighs. The feet are bent underneath the body and to the right side, the position being the same as that of the [sitting] figures found at Hagiar Kim in Malta."² The great size of the thighs recalls the steatopygous type of the Hottentot and the Princess of Punt of Deir el Bahri. The skeleton in the grave was in the usual contracted position. Three other steatopygous figures were found at Naqada: one seated, of dark mud, and two standing, of the light-coloured clay. One figure, however, was found of another and a slighter type, and tattooed, so there must have been two types of women existing together." Professor Petrie has been kind enough to have photographs taken of the two figures in University College, and to allow me to reproduce them. They show a monstrous enlargement of the whole thigh, but it will be noticed that the lateral view shows no superadded gluteal elevation. The figure of the "Queen of Punt" (XVIII Dynasty), or the "Princess" referred to by Professor Petrie, is described and reproduced by Naville in another publication of the

¹ "Naqada and Ballas," by W. M. Flinders Petrie and J. E. Quibell. Certain of the steatopygous figures are represented in Plate VI, figs. 1, 2, 3, 4.

² *i.e.*, The hips and knees are flexed, and both legs, with the feet, brought out underneath the thighs to the right side, as is practised, for example, to-day amongst the natives of Madras.



FIG. 2.

Two predynastic Egyptian clay figures in the museum at University College, London, viewed from the front. In both, the whole of the thigh is monstrously enlarged. Reduced to three-fourths the natural size.

Egyptian Exploration Fund, dealing, amongst other matters, with the temple of Deir el Bahri. The land of Punt, whither the Egyptians repeatedly sent expeditions, is taken to be Somaliland. The temple of Deir el Bahri itself is adjacent to the Tombs of the Kings at Thebes.

On the southern wall of the middle colonnade of the temple (Plate LXIX, loc. cit.) is represented the naval expedition from Egypt to the land of Punt during the reign of Queen Hatshepu. According to Naville's opinion, the Puntites were of the Caucasian type



FIG. 3.

A side view of the left-hand figure shown in the preceding photograph. It will be noticed that there is no superadded gluteal eminence shown, the enlargement involving the thigh generally and uniformly. The inclination of the trunk marks a sitting posture and is not intended to indicate lordosis.

and closely related to the Egyptians; they were not native Africans, but must have come from the opposite coast, probably later than the Egyptians themselves, who belonged to the same race; the appearance of the Puntites, as portrayed at Deir el Bahri, shows that they formed part of the Hamitic stock and that their original home was

Arabia. The sculpture of the Land of Punt, writes Naville, is divided into four rows; there is a shore, and huts built on poles: side by side with the Puntites, and living in huts of the same description, are Negroes: what is brought to the Egyptians by the Negroes (the picture shows commerce in progress) is not quite the same as what is offered by the Puntites; this does not exclude the idea that the Ethiopians had been conquered in a previous campaign: on a stone which the explorer Mariette copied (Mariette, Deir el Bahri), but which has since disappeared, there could be seen the daughter, showing in a less degree the same appearance as the Queen.

It will be seen from an inspection of the figure that the stature of the Queen is about the same as that of her husband. The buttock is very prominent; the lordosis pronounced. Associated with this there is a general obesity; the enlargement of the lower limbs extends to the ankles; and in girth the upper limbs not only greatly exceed those of her husband, but the skin hangs in folds, recalling Speke's description of King Rumanika's fattened wife, in a district not vastly distant. Naville remarks of the steatopygous Queen: "We can thus trace to a very high antiquity this barbarous taste which was adopted by the Puntites, although they were not native Africans." Yet steatopygy is so particularly an African variation that one is led to speculate that the Queen was not herself a Puntite, but an African or the offspring of inter-marriage. Darwin¹ cites Burton to the effect that the Somali men are said to choose their wives by ranging them in a line and by picking her out who projects farthest *a tergo*. The modern Somali, like the ancient, exhibit little Negro mixture; the hair is not twisted; they are Hamitic or Caucasian. Burton's statement cannot imply, then, that steatopygy is common and pronounced amongst the Somali (as a matter of fact it is not), the practice referred to being adopted to select the most of such gluteal preponderance as there is.

In the Ashmolean Museum, Oxford, there are eight Egyptian figures of the predynastic period (Case 1'75). Two are standing; six are sitting, the trunk, except in two, being absent. In each of the six seated figures the thighs, as far as the knees, are abnormally large. In two (one with a headless trunk, the other trunkless) the right leg is shown completely bent and partly to the outer side of the right thigh. It is of importance to note that neither the leg nor the trunk is unnaturally big, indicating that there was not an unusual general obesity. One of the seated figures

¹ "Descent of Man," 1871, chap. xix, p. 346.

(that in which the leg is shown and the headless trunk preserved) is very excellently modelled. The trunk, which is upright and not inclined backwards, is well proportioned without being disfigured by any abnormal obesity or fat above the hips. The thigh is greatly and uniformly enlarged to the knee, but there is no superadded gluteal eminence, the line of the buttock coinciding with that of the back. The same is true



FIG. 4.

The sculpture showing the Queen of Punt, or Somaliland, and her husband, from the temple of Deir el Bahri, referred to in the text. The steatopygia and accompanying lordosis of the Queen are well pronounced; associated with this there is a general obesity, the lower limbs being enlarged as far as the ankles, and the upper limbs being abnormally thick. XVIII Dynasty. From a photograph kindly lent to me by Dr. C. G. Seligmann.

of all six of the sitting figures; the projection of the buttock is either very little pronounced or wanting. The two standing figures are very rudely executed: (1) The whole of the lower limbs and feet are monstrously bulky; the belly obese; the buttocks not particularly prominent, considering the size of the lower limbs: (2) The buttock is prominent; all below it is straight, the lower limb being either unfinished or draped; the upper limb is not abnormally large.

A further Egyptian figure is described in "El Amrah and Abydos," Part I, D. Randall-MacIver. The figure (in Plate L) is from a tomb of the XVIII Dynasty, at Abydos, and is that of an excessively stout and steatopygous woman; there is an accompanying lordosis. The lower limbs are bowed outwards and widely separated. Mr. J. L. Myres, who describes it, observes that, although the face in full view has a negroid look, the profile shows a well-formed nose and little prognathism.

In the British Museum (Egyptian Rooms) there are a certain number of Egyptian female figures, some of which have been adduced as steatopygous, and which I have carefully inspected. With one exception (No. 3), none of these show any proper gluteal eminence, and they cannot be correctly described, therefore, as steatopygous. They exhibit a crural as distinguished from a gluteal lipomatosis. Three of these are wooden dolls:—

(1) Case C, 25.—A well-modelled figure, about 19 cm. in height; slim except for the thighs, which are abnormally large as far as the knees. At the back of the figure the buttocks are accurately indicated by gluteal folds, but are not in the least prominent.

(2) Case C, 26.—A wooden doll almost identical with 25.

(3) Case C, 33.—A wooden figure of a woman nursing an infant, very roughly modelled, the thighs truncated and unfinished. The abdomen is somewhat prominent and each buttock projects in a somewhat pronounced way, but merges above into the back, and below into the thigh; there is no lordosis.

In the Cairo Museum there is a wooden figure, about 8 in. high (regarded by Professor Maspero as a doll), which shows an enlargement of the thighs similar to (1) and (2). Of this Dr. C. G. Seligmann kindly gave me the opportunity of studying two photographs of his own taking. They exhibit a very pronounced enlargement of the whole thigh as far as the knee, but the lateral view shows an absence of any superadded gluteal eminence. Professor Petrie states from an inspection of the photographs that it is probably of the XII Dynasty, not earlier, but that it may be of the XVIII, or possibly as late as the

XXIII—say, between 3000 and 1000 B.C. This figure (like the three preceding, erect) is valuable, as there is no suggestion of its having been executed in a flattened style, which might possibly be urged in the case of Nos. 25 and 26, where the absence of gluteal projection, it might be contended, is attributable to the simplification of the carving.

The other figures in the British Museum which show a similar abnormal enlargement of the thighs are:—

(4) Case 192, 22,906.—A female kneeling on one knee; there is no proper gluteal eminence.

(5) Case 192, 22,905.—An almost identical figure.

(6) Case 192, 22,908.—A female almost identical with the wooden doll, Case C, 26.

(7) Case 192, 2,377.—A female exhibiting a somewhat pronounced enlargement of the thighs, as compared with the generality of similar figures.

(8) Case 192, 32,735.—A female somewhat like 22,908, but the whole figure is more thickly set, and the disproportion of the thighs less marked.

It is to be observed that none of these figures present any gluteal eminence. That they are intended to represent the feature they do would appear from the fact that they are exceptional; the many other female figures are slim in the lower limbs. In addition to these wooden figures there is an ivory one from the Archaic period, viz.:—

(9) Case L, 173.—A female, of which the feet and head are absent. The whole of each lower limb, including the portion of the legs remaining, is unusually large, but there is no steatopygy, and the figure, which is excellently executed, represents a condition of general obesity.

(10) Case L, 42.—A small, flattened, badly executed female with short limbs, the lower being abnormally broad. There is no steatopygy, but the thinness of the material from which the figure is made would render it impossible to show such a feature had it been present.

The craniological evidence as to the existence of a negroid population in ancient Egypt has been analysed by Arthur Thomson and D. Randall-MacIver in "The Ancient Races of the Thebaid," 1905. These authors observe that the evidence shows clearly that the population of Upper Egypt, even in the earliest days, was made up of diverse elements. The facial skeleton shows two distinct groups, one negroid and the other non-negroid; during the predynastic periods the negroids were the social equals of the others. As regards the negroid element, they remark that

the immediate proximity of Nubia naturally suggests an influence from that quarter. With regard to the non-negroid stock, this might have been affiliated to the Berber-Lybian, to some other Mediterranean, or to a Semitic stock. No definite pronouncement can be made.

Professor Petrie comments on the discoveries at Naqada and Ballas (*loc. cit.*, p. 34) as follows: "In the graves at both Ballas and Naqada were found several figures modelled in whitish marly clay or in Nile mud. These represent a race which is otherwise not found in Egypt. The steatopygy and the characteristic lumbar curve in the standing figures seem to connect this with the well-known Hottentot type. At first sight it may seem strange to adopt so distant a connexion, but it appears that this race has gradually receded before the pressure of higher races. This form is shown in two ivory carvings found in the cavern of Brassempouy in the south-west of France, about thirty miles from the Bay of Biscay and fifty miles from the Pyrénées.¹ The figures prove that a Hottentot type existed in that region at a period which is equal to that of Solutré, that is to say, the second of the four periods of the Palæolithic age. In neither Brassempouy nor among the 'New Race'² is this type the only one; a slender European type is associated with it. We may next note this same steatopygous race in Malta. The seven seated figures carved in limestone which were found in the rude stone temple of Hagiar Kim (Adams, 'Malta,' vol. vii, 1) are very closely like those in the graves of the New Race. There is the same monstrous thickness of the legs, and the same attitude of sitting on the ground with the feet both turned out to the right-hand side, an attitude never shown in Egyptian figures. Then to the south this type is shown by the Queen of Punt or Somaliland in the XVIII Dynasty on the sculptures of Deir el Bahri; and in modern times it is only known in the South of Africa. There is thus a series of five regions in which the steatopygous race appears, and which lie apparently from north to south in the order of successive dates of the remains."³

ANCIENT CRETAN AND GRECIAN FIGURES.

Of all the ancient figures extant the most valuable, in my estimation, as establishing the existence of true steatopygy, are two in the Ashmolean

¹ Piette, in "L'Anthropologie," 1895, vi, 2, 129-151.

This is the term used by Professor Petrie as meaning the predynastic race which he regards as of Lybian origin.

² *i.e.*, Brassempouy, Egypt in the "New Race," Malta; Punt, in the XVIII Dynasty, South Africa.

Museum, Oxford, from the Palace Hill, Cnossus, Crete. These are two sitting idols in black, hand-polished ware with incised whitened decoration; the upper part of the trunk is absent. In neither are the thighs at all enlarged; their posterior surface, indeed, is slightly concave, instead of being convex or flat. But projecting directly backwards, far beyond the line of the back in each, are two very prominent gluteal eminences parted in the mid-line by a wide cleft. There is a third steatopygous figure in the Ashmolean Museum, from Cnossus, a marble idol, No. '09408. The figure is headless and the chief part of the lower limbs wanting, but it is probably sitting. The trunk and arms are slim, the buttocks very voluminous, projecting behind the line of the back, and separated mesially by a cleft; the swelling extends laterally, the trunk appearing to arise from a huge circular cushion.

A further ancient Cretan steatopygous figure (headless) has recently been discovered by Angelo Mosso during the excavations at Phæstos.¹ The figure is in hard unbaked clay, 6 cm. high, with the thighs truncated. Enough of the thigh is represented to suggest that it was abnormally large, but there is in addition a superadded gluteal eminence of moderate size; the abdomen is large and pendulous. In his work "*Idoli femminili e figure di animali nell' età neolitica*" the same author refers to similar female idols found in Italy.

A comparison of the pottery found in ancient Egypt with that found in ancient Crete proves that commercial relations between the two had been long established: the stone vases brought into the island correspond chronologically with those produced in Egypt during the first Egyptian dynasties. And, conversely, Cretan vases discovered by Petrie at Abydos are identical with those found in Crete (at Cnossus) at the depth where the remains of the Bronze Age begin. The strata in the Court of Cnossus at a depth of 5 metres correspond chronologically with the first Egyptian dynasties. Below this is a stratum $6\frac{1}{2}$ metres thick, in which only stone implements are found, before the virgin soil is reached. The clay figure from Phæstos was found in the Neolithic stratum, and would thus go back to the predynastic Egyptian period, like those from the graves at Naqada and Ballas.

From ancient Greece there is in the Ashmolean Museum (Case 1'46) a primitive female image in Pentelic marble, from Patesia, near Athens. It is ill-executed; the chest is abnormally large, the buttocks and hips pronounced; the lower limbs are truncated. Perrot and Chipiez ("Art

¹ "Palaces of Crete," by Angelo Mosso, 1906.

in Primitive Greece," vol. ii, fig. 330) represent a poorly executed female idol of white limestone, from the neighbourhood of Sparta. The figure, which is standing, shows a generally obese woman, with somewhat voluminous buttocks; the lower limbs are enlarged to the ankle.

MALTA.

Of the sitting figures carved in limestone at Hagiar Kim (Adams, "Malta," vol. vii, 1), already referred to, there are photographs in the Ashmolean Museum (Case 1, M). They are very striking and satisfactorily executed. The lateral view of one shows a huge thigh which merges into and culminates in an enormously voluminous buttock projecting far behind the line of the back. The legs are notably fat, and so are the upper limbs, there being an evident general obesity. The enlargement of the legs below the enlarged thighs appears in all.

THE BRASSEMPOUY FIGURES (SOUTH-WEST OF FRANCE).

The ivory figures (alluded to by Professor Petrie) from Brassempouy are carefully described and figured by their discoverer (Piette, loc. cit.), and I may here notice them somewhat in detail. The chief is what the author distinguishes as the Brassempouy Venus. There is a general enlargement of the thighs, but no superadded gluteal eminence, and he expresses a doubt whether the condition is identical with that of the Bushmen. This is the figure taken to show, also, an elongation of the nymphæ "terminated by a vulviform appendix." An excellent full-sized figure of the elongated and apposed labia minora, or "Hottentot apron," is given in Billroth's "Frauenkrankheiten," vol. iii; the nymphæ project for a distance of 4.5 cm. beyond the lower limits of the labia majora. Piette's expression is ambiguous; the "termination" apparently refers to the anatomical origin of the nymphæ, which normally protrude from between the slightly parted labia majora. The specimen is somewhat damaged, and without an inspection of the actual object one may be excused from passing an opinion upon such a detail. In a second ivory figure ("Le Manche de poignard" de Brassempouy) there is a marked lateral prominence of the hips as viewed from the front, but neither the lateral nor the posterior view shows any steatopygy. A third figure (Mas-d'Azil) is very badly executed from a large incisor tooth. The lower part of the figure, including the hips and buttocks, is unfinished and represented by the original part of the tooth.

Nothing can be deduced from it beyond the presence of lateral folds (of fat) above the position of the hips. The nose is large and rounded; there is no prognathism; the upper jaw considerably overhangs the lower. The fourth and last figure ("Femine au renne") shows somewhat thick thighs, but no proper steatopygy. The other statuettes of Brassempouy (like the Egyptian) represent a slender type of figure, showing that the obese form was not universal, or that a mixed people occupied the regions in question.

ANALYSIS OF THE FOREGOING OBSERVATIONS.

The modern Hottentot-Bushman steatopygy is of a well-defined character, and the presence of the hump is not necessarily associated with any enlargement of the rest of the thigh. So it is always represented by the people themselves, as already noticed. And when the thigh is enlarged it is of importance to observe that the hump is still well differentiated, without being merged into the rest of the enlargement. There is in the Dermatological Collection of the Royal College of Surgeons the photograph of a Bushwoman in whom, associated with a marked gluteal hump, there is an enlargement of the whole of both thighs, particularly over the hips, which brings out this point; the rest of the body is slim. This form of local obesity, or steatopygy in its pure, unmixed kind, indeed, is so well differentiated that it might be distinguished as a *Gluteal lipomatosis*. For it will be granted that, for the sake of accuracy, the term "steatopygy" should not be used to cover enlargements that are lateral only, or conditions where the thighs alone are large. I have already cited Sir Harry Johnston's observation that amongst the Forest Negroes of Congoland a lateral enlargement of the hips occurs, which is unassociated with rearward projection or marked prominence of the buttock. This might be distinguished as *Coxal lipomatosis*. It is apparently the form represented in one of the Brassempouy statuettes (figs. 1, 1A, 1B, "Le Manche de poignard," loc. cit.).

Amongst the many ancient "steatopygous" figures, whether Egyptian, Cretan, Grecian, Maltese, Italian, or French, there are only two, so far as my examination goes, that accurately reproduce the proper Hottentot-Bushman form, or true gluteal lipomatosis. And these are of great value as establishing the occurrence of this type, and placing beyond doubt what might otherwise be questioned—viz., the existence of Bushman steatopygy in ancient times in a locality so far removed as Crete from its present distribution in South Africa. These are the two sitting

figures from Cnossus to which I have already referred, and which show the well-defined and prominent gluteal gibbosity unassociated with, or obscured by, any enlargement of the thighs. Some of the so-called steatopygous figures show an abnormal size of the thighs, but without any gluteal eminence. The wooden dolls and certain other figures in the British Museum previously alluded to are of this kind, and so is the "Brassempouy Venus." That the enlargement is not intended to represent merely the normal fullness of the female thigh would appear from the fact that such figures are exceptional, for other dolls of similar size and of as excellent a technical quality do not exhibit it. This enlargement cannot properly be classed as steatopygous; it might be distinguished as *Crural lipomatosis*.

Other ancient figures exhibit a general as distinguished from such local obesity, though I know of none such in which the condition is so highly pronounced as to merit the current pathological name of general lipomatosis. In these (*e.g.*, the ivory archaic female figure in the British Museum, Case L, 173) the legs and trunk are abnormally massive as well as the thighs, without the latter or the buttocks being disproportionately so.

In the predynastic Egyptian clay and Nile-mud figures the most striking feature is the enlargement of the thighs; no steatopygy is shown in any, unassociated with the extraordinary crural enlargement. If the rude art of the modern Bushman is sufficient to enable him to depict accurately the local accumulation on the buttocks of his race, we may assume that the predynastic art of Egypt, which was much higher, would in a fair way speak the truth when representing the monstrous enlargement as regularly involving the whole of the thigh. It is unnecessary to point out that the representation cannot be referred to elephantiasis, seeing that in the latter the swelling of the lower limb proceeds from the foot upwards, and the form of the knee becomes obliterated. Nor is the crural enlargement merely part of a general obesity, since when the trunk and legs of such figures happen to be preserved they present no unnatural volume.

It will be obvious, however, that whilst a local lipomatosis is something *per se*, a general obesity may be superadded to it. In such cases the disproportion will still be maintained, for the local enlargement will be still further exaggerated. The monstrous enlargement of the thigh reaches a maximum in the seated limestone figures at Hagiar Kim, Malta; and these exhibit, in addition, an enlargement of the legs below the knee and of the trunk and upper limbs, and such a rearward

projection, that it must be named *steatopygous* and taken as constituting part of the local lipomatosis. The Queen of Punt also, I think, illustrates such a combination of a local and a general obesity.

All the predynastic Egyptian figures exhibit a crural lipomatosis, but the gluteal or proper *steatopygous* eminence varies. In the sitting figures in the Ashmolean Museum there is very little or no rearward projection



FIG. 5.

A Bushwoman in whom *steatopygia* is associated with enlargement of both thighs as far as the knees. It will be noticed that the gluteal eminence is not confused with the enlargement of the thighs, but is superadded to it. From a photograph in the Museum of the Royal College of Surgeons.

beyond the line of the back. In that figured from University College, London, in the present communication, and in that figured by Professor Petrie in Naqada and Ballas (Plate VI, fig. 2, the original of which is

in the Cairo Museum ?), there is some amount of gluteal projection, but in comparison with the great size of the thigh it is certainly not pronounced. The lipomatosis is particularly a crural one in which the enlargement of the buttock is not the special feature, but rather a variable accompaniment of that of the thigh. In this it differs from the proper Hottentot-Bushman type, which is first and foremost gluteal and is quite independent of any enlargement of the thigh itself. The Egyptian form is a crural or gluteo-crural; the Bushman, a gluteal or cruro-gluteal.

That the form in question is a variation now obsolete or extinct, Schweinfurth's statements in regard to the Central African Nilotic Bongo women would make it difficult to substantiate. Variations in the disposition of local lipomatosis we must be prepared to allow. In the fat-tailed *Dasyures* of Central Australia the whole tail is, in some varieties, enlarged; in others only the proximal half. There are, again, the familiar examples of the one-humped and two-humped camel, and the variations presented by different breeds of fat-tailed sheep, which I shall have hereafter to describe.

The deductions of Professor Petrie (previously cited) may need some modification in detail, but broadly they must be true. For if the different varieties of lipomatosis shown in the several ancient figures are not all referable to the Bushman type, they are nevertheless African. And so far as Egypt goes the craniological data prove the co-existence of a negroid and a non-negroid population, even in predynastic times.

And lastly, having regard to the proximity of the different localities in which these figures have been discovered to the African continent, it would seem simpler to think of "steatopygous" people as having migrated from this continent to neighbouring parts, than that Africa was peopled by such from a European source.

The accentuation and transmission of steatopygy may, as Darwin points out, be attributed to sexual selection, seeing that the conformation is admired by the people among whom it occurs. One must not forget also that its persistence would tend to be brought about by natural selection as well. For amongst an uncivilized, wandering people this local accumulation of fat would be of decided advantage as a food-reserve in enabling the individuals so favoured to survive through an adverse extremity.

The Humps in the Camel and Zebu.

These steatopygous accumulations we must view, indeed, as strictly analogous to the dorsal store of fat, say, on the back of the Camel. In *Camelus dromedarius* (Africa, Asia Minor) the eminence is single; in *Camelus bactrianus* (Asia, particularly Central Asia, north of the Himalayas) it is double. In the well-nourished animal it is large, elastic, and erect; but in the ill-fed, starved, or diseased, it hangs down and may be scarcely discoverable. Mr. R. I. Pocock tells me that such variations are even noticed in the camels living in the gardens of the Zoological Society. Owen¹ remarks in this connexion that the camels are able to endure unusual fasts by reabsorbing these accumulations, concluding their journeys across the desert with the special stores of fat much reduced. There is a portion of the hump of *Camelus dromedarius* in the College Museum (Physiological Series, Specimen No. 1840) which displays its gross structure, and of which I have made a microscopic examination. The divided surface is almost homogeneous, opaque, and of a pale yellow colour, with no obvious intersecting septa of connective tissue. The uniformity of the section is broken only by the presence of a certain number of short, fine streaks of connective tissue devoid of any definite arrangement. Histological examination shows the tissue to be of the common adipose kind, its cells being unilocular, as in ordinary fat. No muscle fibres are present, the scanty broken lines in the fat consisting of wavy, fibrous tissue with fine, curling, intermingled elastic fibres.

In both *Camelus bactrianus* and *Camelus dromedarius* the hump is present at birth, but in a disproportionally diminutive degree. In the *Camelus bactrianus* born in the gardens of the Zoological Society in April, 1909, the humps were represented by two folds of skin. And in a specimen of foetal *Camelus dromedarius* which Captain S. S. Flower was good enough to send me from Cairo the back presents a long, tegumentary fold, which I may describe more in detail. The skin of this foetus is everywhere well covered with white, somewhat curly hair. In accurate sagittal section carried through the spinal column and suprajacent structures the longitudinal extent of the eminence is 20 cm.; it rises quite gradually from the general level; its thickness from side to side is about 3 cm., and its extreme vertical measurement is but 2 cm.

¹ "Lectures on Comparative Anatomy," 1866, iii, p. 783.

The corium over it is distinctly thicker than elsewhere; below this the eminence consists of a tough, pale-yellow tissue. I examined it microscopically by means of coronal sections made to include the cartilaginous epiphysis of the vertebral spine. On the cartilage there is a thick perichondrium. Between the deep fibrous structures lying on and incorporated with the perichondrium and the well-defined corium (in which hairs are abundantly implanted) the whole of the tissue consists of well-developed fat of the common unilocular-celled kind. The fat is traversed by somewhat narrow bundles of connective tissue, but of the two the adipose is in excess. There is no striated muscle fibre intermingled with the fat.

Geoffroy Saint-Hilaire and F. Cuvier state¹ that in both species of Camel the humps are present at birth. In the degree described, this is true.

After the removal of storage fat, its utilization, according to the hypothesis of Leathes, is brought about through the agency of the liver, in which such fat is transformed by a process of "desaturation" into fat-like bodies or lipoids, which are thence distributed in a utilizable form to the more important organs of the body, including, of course, the liver itself. The iodine value of the fatty acids extracted from the liver is normally much higher than that of fat from the subcutaneous tissue—*i.e.*, the fatty acids are acids less "saturated" than the normal oleic series. [The unsaturated acid is one in which the carbon atoms are joined together by more than a single linkage; in a saturated acid one carbon atom is joined to another by one linkage only, the others being separately satisfied by atoms other than carbon. A saturated fatty acid can only change its state by substitution; an unsaturated one can change by "addition," the former being the more stable.]

The recent observation of P. Hartley and A. Mavrogordato²—that, when the amount of fat in the liver is greater than that commonly found in health, the iodine value of the acids of this fat approximates to those of adipose connective tissue—has served as one of the foundations for the hypothesis of Leathes already referred to. Leathes, moreover, has pointed out that, whilst the amount of fat in the heart and kidney is fairly constant, that in the liver varies, indicating again that in the latter organ a process of transformation takes place. When

¹ "Histoire naturelle des Mammifères."

² *Journal of Pathology*, Camb., 1908, xii, p. 371.

there is an excess of fat in the liver the iodine value of the fatty acids approximates to that of the connective tissue, either because the transportation has been so excessive that time has not allowed of the conversion to occur, or because the liver is, for some reason, incapable of effecting it in full. The lipoids themselves, of which the most definitely known is lecithin, contain nitrogen (phosphatide lipoids).

The hump on the withers of the Zebu (*Bos indicus*), numerous breeds of which are distributed over India and Africa, is an example of a somewhat different kind. Some of the breeds, *e.g.*, the Gynsee cattle, are quite diminutive, yet they preserve the characteristic hump, of course in a proportionately reduced size.

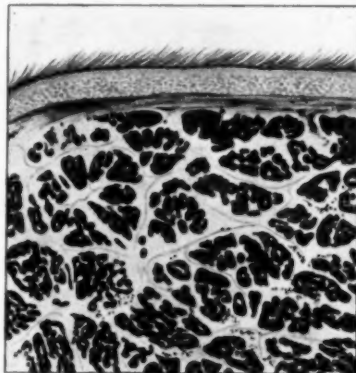


FIG. 6.

A portion of the hump on the withers of the zebu, including the overlying skin. It consists chiefly of coarse bundles of muscular tissue, a greatly exaggerated growth of the panniculus carnosus; between the muscular bundles there lies a smaller amount of fat. (Museum of the Royal College of Surgeons.) Natural size.

Through the kindness of Mr. R. I. Pocock, the Superintendent, and Mr. H. G. Plimner, I have had the opportunity of examining the hump from a zebu cow which died in the gardens of the Zoological Society. The animal came to the gardens with the history of a fall, and died of pleurisy and pericarditis; after death some old blood-clot was found in the muscles of the right side of the back, but none of the bones had been fractured. The cow had been ill in the gardens for three weeks.

The hump is a firm, hemi-elliptical mass, 9 in. in its longer diameter at the base of attachment, and about 6 in. from side to side. The overlying skin is not particularly adherent, and admits of being stripped from the eminence, with which it is connected by a thin layer of subcutaneous fat. As readily seen by the naked eye, in the sagittal section carried through it from end to end, it consists throughout of coarse, reddish-brown bundles of muscular tissue, separated by narrower lines of fat. The muscular substance is presumably a vastly overgrown portion of the panniculus carnosus, and of the two constituents, which are uniformly intermixed throughout, the muscular slightly predominates over the fat. The muscular tissue itself is not inserted into the corium, so that a clean separation can be made on dissection without division of the muscle fibre. Histological examination confirms the naked-eye conclusion. The fat ramifying between the bundles of muscle (which is of the striated kind) is composed of unilocular cells of the common type. The proportion of fat in the hump of the zebu varies, however. The small amount in the case just cited must be attributed in part to the wasting caused by the animal's illness.

In a second zebu, which was killed at the Gardens for the general food supply, the hump, which was about 20 in. along its base and 10 in. from side to side, consisted much more extensively of fat. The proportion of fat varied in different places, and in some there was no more than in the first specimen, but the fasciculi of muscle elsewhere became more and more disparted by fat until the latter exceeded the former in amount. This particular animal was notably fat.

The hump of the zebu differs, therefore, from that of the camel in the large proportion of muscle it contains, and although to a certain extent a fat reserve, it is not so to the degree it is in the camel; and yet it may be viewed as a reserve, but one of protein as well as of fat; no other function, at least, can at present be assigned to it. In India the hump is regarded as a delicacy and is salted and boiled like beef.

In a Gynce calf, born in the gardens of the Zoological Society in December, 1908, I found the eminence well pronounced at the age of seven months, and relatively about as large as in the adult. The keeper informs me that the hump was present at birth, and that this is always the case. In the ordinary wear and tear of muscle the products are excreted in the urine as nitrogenous waste (? creatinin), but under exceptional circumstances muscular tissue may be utilized for maintaining or building up the protein constituent of protoplasm. In starvation the organs whose function is essential to life not only draw upon the fat as a

reserve supply of energy, but also upon the voluntary muscles, which are sacrificed for the upkeep of the heart and other viscera; from the products of their disintegration protein, in short, is rebuilt.

This is taken to happen, again, in the case of the salmon, the disintegrated products of the muscular tissue of which are utilized for the growth of the ovary and testis, which takes place when the fish leaves the sea for spawning. In the salmon, to which I will again refer, there is an additional source of energy in the intramuscular storage of fat; but fat, being non-nitrogenous, is only capable of supplying energy to functioning protoplasm, whether of gland-cell, nerve-cell, or muscle: it is incapable of building up nitrogenous protein.

The zebu hump is a mixed reserve of protein and fat, and in the event of extreme starvation it would, after giving up its fat, supply the constituents of protein to the organs whose function is essential to life.

The hump of the Bison (*Bison americanus*; *Bison europæus*) and that of the Yak (*Bos grunniens*) are due to the elevation of the withers (or the length of the spinous processes of the vertebræ), and so lie outside the subject under consideration.

The prominent "crest" which bears the mane in the uncastrated Horse, or stallion, is so largely composed of fibrous tissue that it cannot be classed as a fat store. Its development is lessened by castration, when carried out in the young animal, the neck of the gelding coming to resemble that of the mare. And the same exclusion must be made in regard to the still more conspicuous crest in the Ass. In this animal the fibro-fatty tissue forms two long, prominent, subcutaneous elevations, one on either side of the neck below the mane. Although these shrink during emaciation, they do so in no pronounced degree.

Tumour-like Formations of Fat in the Tail.

The local storage of fat, however, is not confined to such dorsal humps. In another group of cases it takes place in the tail. In certain of the *Dasyuridæ*, a group of the smaller marsupials, the tail is conspicuously large from the accumulation of fat, in some species at the base only, in others throughout. Figures of these animals are given by Baldwin Spencer in the report of the work of the "Horn Scientific Expedition to Central Australia," Part II, Zoology, 1896. In *Phascogale cristicaudata* the whole of the tail is thick. In *Phascogale Macdonnellensis* the thickening, which is highly pronounced, is confined to the basal half; in *Sminthopsis larapinta* the condition

is much the same. A specimen of one of the two latter has been recently acquired by the College; on one side of the enlarged portion of the tail the skin has been reflected to display the fat beneath it. Nothing is known here as to whether any periodical diminution or augmentation occurs in connexion with winter (hibernation) or summer sleep (æstivation).¹ It is pointed out in the report mentioned that the region in question is arid and inhabitable only by animals able to travel long distances, or by others which are chiefly burrowing and able to live with little water and to feed upon insects, such as ants, &c. This would favour the supposition that such accumulations of fat may serve

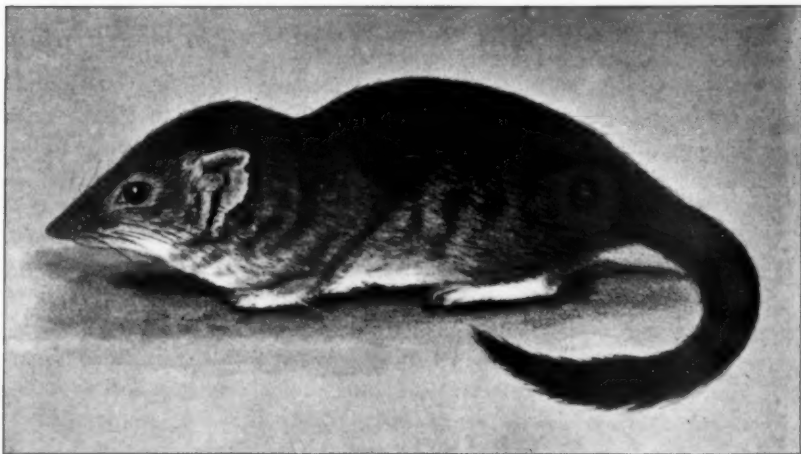


FIG. 7.

Phascologale cristicaudata, showing the large size of the tail, due to the accumulation of fat. (After Baldwin Spencer.)

as food-stores. And this probability is supported by the fact that in the case of the fat-tailed gerbille (*Pachyuromys Duprasi*, Egypt), of which examples have been kept in the gardens of the Zoological Society, the tail has been observed to attenuate when the animal for any cause emaciates; so Mr. R. I. Pocock informs me.

¹ The Tanreos (*Centitida*) of Madagascar are stated to have a summer sleep, like the winter sleep of the hedgehog, which animal they much resemble in character. This æstivation was observed during the summer of 1909 in the Tanreos received in the Gardens of the Zoological Society.

The expanded tail of the Ornithorhyncus (*Ornithorhyncus anatinus*, South Australia and Tasmania) owes its breadth and thickness to an accumulation of subcutaneous fat, in the midst of which the caudal



FIG. 8.

Phascogale Macdonnellensis, showing the thickening of the basal half of the tail from the accumulation of adipose tissue. (After Baldwin Spencer.)

vertebræ, thinly ensheathed with muscle, are concealed. In the particular specimen which I have examined the length of the tail as measured along its slightly concave inferior aspect, from the cloacal orifice to the tip, is 11 cm.; its breadth immediately beyond the same orifice is 8 cm.; its maximum thickens 3 cm., the subcutaneous fat itself being about 0.7 cm. in thickness, either ventrally or dorsally to the muscular and osseous centre. The subcutaneous fat over the rest of the body is of no unusual thickness. Histological examination of this fat shows it to be adipose tissue of the common kind, the component cells being large, spherical, and unilocular.

But much more striking illustrations of normal or physiological depositions of fat are afforded by the breeds of sheep which, in accordance with the position of the accumulations, are known as fat-tailed and fat-rumped. Excellent figures of two varieties of such are given by Prichard in his "*Natural History of Man*" (4th ed., i, 1855); and there is a typical example of each of these two, prepared and exhibited in the British Museum (Natural History) at South Kensington. In one the tail is of a length to reach the ground and is throughout thickened; in the other the tail is rudimentary and the fat lies not in the tail, but on the buttock. These several varieties of sheep are spread more especially over extensive areas of the East—Arabia, Syria, Palestine, Persia, Turkestan, and China. And here it is a matter of direct observation that the fat-tailed sheep of the Kirghiz tribes (north-east of the Caspian Sea) when transferred more northwards into Siberia do not retain their peculiar character. Darwin¹ cites Pallas and also Ermon ("*Travels in Siberia*," English translation, i, p. 228) to the effect that in the Kirghizian sheep, when bred for a few generations in Russia, the mass of fat on the tail dwindles away. "The dry and bitter herbage of the Steppes is unfavourable to the growth of fat, and they lose the mass of adipose matter" (Prichard, loc. cit.). The disappearance under the above circumstances must be attributed to the absorption of the fat accumulated, and to the cessation of further storage brought about by the conditions of such an unfavourable environment.

But another region in which fat-tailed sheep occur is in South Africa. In the Museum of the Royal College of Surgeons there is the tail from one of these sheep, obtained by the indefatigable industry of

¹ "*Variations in Animals and Plants under Domestication*," 1868, i, p. 93.

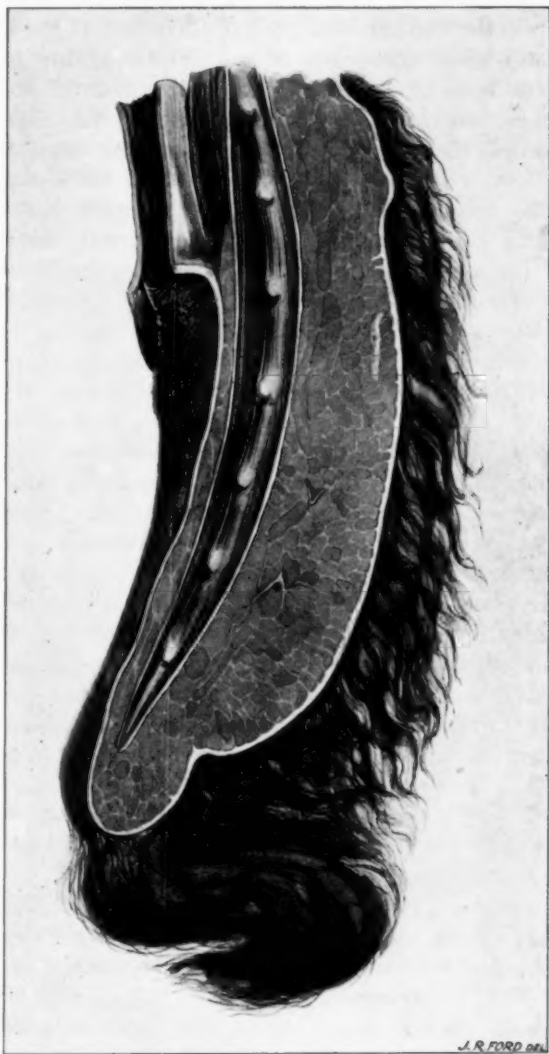


FIG. 9.

The tail of a short, broad-tailed sheep from the Cape, divided to the left of the mid-line. The accumulation of fat is confined to the subcutaneous tissue of the proximal portion. The muscles upon the caudal vertebrae have been exposed; these are normal in colour and consistence. The termination of the gut is shown at the highest part of the specimen. (Specimen No. 2074, Physiological Series, Museum of the Royal College of Surgeons.) Reduced to one-half the natural size.

Hunter. It is catalogued (No. 2074, Physiological Series) as "the tail of a sheep from the Cape of Good Hope." The tail, of which a vertical section has been made to one side of the mid-line, is 6 in. in its entire breadth, at the level of the anus; it is $2\frac{1}{2}$ in. thick. For a length of 5 in. it almost maintains its breadth, but beyond this spade-like proximal portion the organ abruptly assumes the usual form, being for another 6 in. cylindrical and slender, though curled up instead of pendent. The broadened proximal segment is quite hairless on the anterior or deep aspect, the skin over it being directly continuous with that around the anus. The muscular tissue upon the caudal vertebræ is well developed and of normal colour. The accumulation of fat is practically limited to the posterior and lateral aspects of the vertebræ and is firmly connected with the skin, the fat being strictly subcutaneous. Histologically, the fat, I find, presents no peculiarities; it consists of large unilocular cells. There are, at the present time, living examples of such short, broad-tailed sheep in the gardens of the Zoological Society. The animals were received with other cattle from India and are catalogued as Dumba sheep. The tail, which I have had the opportunity of examining in the living animal, is much as in Hunter's specimen, but so much broader at the base that it completely overlaps the buttocks, so completely, indeed, that without examination the buttocks themselves might be taken as the seats of the enlargement, especially as there is appended to the end of the broad basal portion of the tail a diminutive cylindrical stump representing the terminal, unenlarged part of the member. The sheep might be mistaken, indeed, without examination, for the fat-rumped variety to which I will presently refer. The broad portion of the tail is bilobed at the lower border. In Hunter's specimen this subdivision is absent and the tail is less widely expanded, both differences being obviously due to a lesser degree of local obesity.

This form of short, broad-tailed sheep is one of those also met with at the present day in the East—in Arabia, Syria, and Palestine. It is one of those referred to by Herodotus,¹ and of which sculptured bas-reliefs have been found among the ruins of Persepolis, the ancient capital of Persia. In speaking of Arabia, the historian writes: "There are also in Arabia two kinds of sheep worthy of admiration, the like of which is nowhere else to be seen. The one kind has long tails, not less than three cubits in length, which, if they were allowed to trail on the

¹ Book III, par. 113, Rawlinson's translation, 1875.

ground, would be bruised and fall into sores. As it is, all the shepherds know enough of carpentry to make little trucks for their sheep's tails. The trucks are placed under the tails, each sheep having one to himself, and the tails are then tied down upon them. The other kind has a broad tail, which is sometimes a cubit [18 in.] across." And it has been pointed out by more than one Biblical commentator that the Levitical injunctions appertaining to the sacrifice of the peace-offering (Leviticus, iii, 9, and Exodus, xxix, 22) relate to fat-tailed sheep of



FIG. 10.

A short, broad-tailed Dumba sheep. Photographed from life from an animal in the gardens of the Zoological Society. A piece of canvas has been stretched beneath the tail, which completely overlaps the buttocks. From the lower border of the bilobed enlargement there projects the short terminal cylindrical segment of the tail, obscured by the wool.

this or of the long-tailed variety, and that the word adopted in the English version ("rump") should refer to the tail, the Hebrew term being identical with the Arabic, which the Arabs apply to the tail. In

the directions detailing the sacrifice of the goat (Leviticus, iii, 12, *et seq.*) it is a significant confirmation of this that, whilst the fat of the belly and internal parts is referred to, no mention is made of the "rump." In the Jewish bible (the translation by Dr. A. Benisch, under the superintendence of the Chief Rabbi) the passage from Exodus runs: "Thou shalt take of the ram the fat and the fat tail," &c.; and from Leviticus: ". . . the fat thereof, and the whole fat tail shall he take off hard by the backbone," &c.

In the "*Histoire naturelle des Mammifères*" (Geoffroy Saint-Hilaire et Frédéric Cuvier, tome 1) there is a figure (from behind) of a short, broad-tailed sheep in which the accumulation of fat reaches a maximum. The lateral masses project beyond the buttock at the sides, and the median rudiment of the tail is likewise enlarged so as to form a third lobe which hangs for a short way between the other two. The figure is reduced to one-seventh of the natural size: when the actual breadth of the tail is calculated it comes, I find, to 18 in.—exactly that given by Herodotus as the breadth in the Arabian fat-tailed sheep of his day, viz., a cubit.

There are, it will thus appear, variations in the form assumed by the tails of fat-tailed sheep, as there are in the *Dasyures* already referred to, where the adipose enlargement may be generalized or confined to the basal portion of the tail. As already mentioned, besides that form in which the proximal portion is broadened by fat, the terminal moiety being narrow and short, the whole tail may be enlarged and reach the ground. In the prepared specimen from the Cape, exhibited in the British Museum (Natural History), South Kensington, the massive tail tapers from the base to the ground. Although broadest and thickest at the root, it is not, in this particular example, expanded to the degree it is in the other broad-tailed variety, which for distinction may be called short, for the sides of the buttocks are not concealed as the animal is viewed from behind. On the deep or anterior aspect the tail is, throughout its length, hairless and pink, as it is in the shorter-tailed form. In order to keep the trailing end of such a tail from abrasion, it is customary, as is well known, to fix below it a light truck of wood on small wheels. As will appear from the quotation from Herodotus already given, both of these forms of fat-tailed sheep existed in his day, and the practice of using a truck was in vogue even at that time. Similar natural variations in the length of the tail are met with also among British breeds of sheep, in which the tail may reach the ground or not extend below the hocks. But, apart from such accumulations

of fat in the tail, the fat in another variety of sheep is confined to the buttock—the sheep is fat-rumped instead of fat-tailed. In the fat-rumped sheep (*Ovis steatopyga* of Pallas) the tail is short and rudimentary, or even absent, and there is a prominent, flattened hemispherical accumulation of fat upon each of the buttocks. In the broad-tailed sheep the anus is overlapped and concealed by the expanse of tail; in the fat-rumped sheep the anus lies between the gluteal eminences of fat, which are, nevertheless, to some degree pendent, or

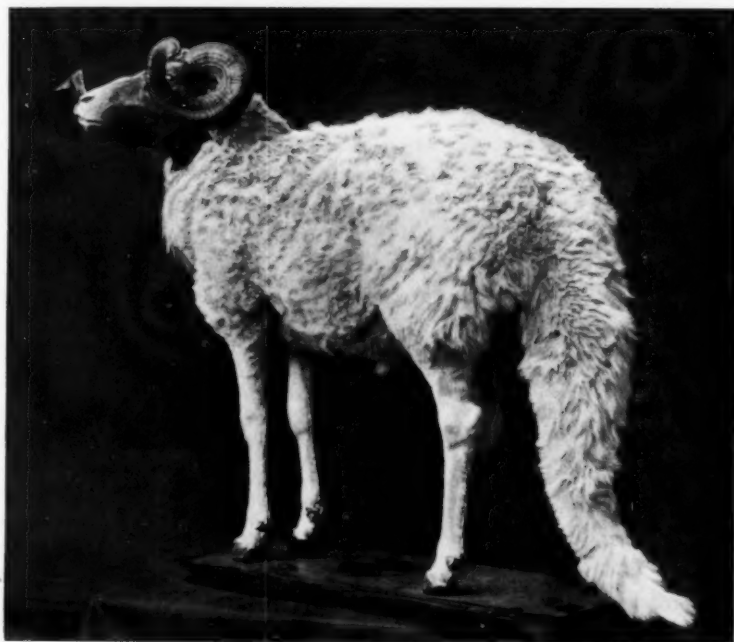


FIG. 11.

Long, fat-tailed sheep from the Cape. Photographed from a prepared specimen in the British Museum (Natural History), South Kensington.

separable at their lower borders from the parts beneath; the perineum is unencumbered. The example shown at the British Museum is from Hedjaz, Arabia, and of this I append a photograph. Such sheep are found in Persia and Arabia, and many parts of Central Asia, in the

countries to the east and south of the Caspian Sea, and they are found also in Africa, chiefly in the north-east.

The earlier extension of fat-tailed and fat-rumped sheep to the African continent offers, of course, no geographical difficulty, any more than the migration of Caucasian or Hamitic races to the same continent by the isthmus of Suez or across the Red Sea, &c., the sheep having probably accompanied man in his peregrinations, in the same way that the camel, originally a native of Asia, was thence introduced into Africa.

Living examples of fat-rumped, or steatopygous sheep are in the possession of Sir Claud Alexander, of Faygate Wood, Sussex, who has been good enough to furnish me with the following observations: In the summer of 1907 he obtained a ram, three ewes, and two ewe lambs. All were in very poor condition, but they improved rapidly, and as they did so their rumps increased, but not in the same ratio as the humps of Indian oxen had been observed to do by Sir Claud Alexander, under similar circumstances. Each of two of the ewes afterwards had a lamb. These two lambs, which were tailless, presented no sign of the fat rump; the latter, however, became quite conspicuous in about three days, and increased with remarkable rapidity.

It will appear, from the foregoing data, that when the tail is absent or rudimentary, the fat accumulates over the buttock; if there is a tail, the accumulation advances into it and the sheep becomes fat-tailed. That the two conditions are closely related is well shown by the examination of a young lamb which Sir Claud Alexander kindly sent to me from his fat-rumped stock. In this specimen (now in the Royal College of Surgeons) there is no trace of tail, but on each of the buttocks there is a prominent elevation of fat. The central part of the lower border of each lobe, however, is sufficiently pendent to project 5 cm. below the level of the anus. The bases of the two lobes are united for a distance of 3 cm. on the deep aspect immediately over the anus by a thin fold or commissure of skin; the anterior surface of all the pendent parts is hairless. As seen in the sagittal section, the vertebræ terminate at the bottom of the cleft, and do not project beyond the level of the anal aperture. It is evident, here, that it only needs the presence of a tail to conjoin the lobes and allow of their still further extension, and the sheep would be fat-tailed in place of being fat-rumped, the fat in the latter variety being fixed to the higher part of the buttock at the site where the root of the tail would lie. And this is really the condition in the tailed hybrids raised by Sir Claud Alexander from these fat-rumped and ordinary sheep.

Amongst Bovines, in some strains of shorthorns and other pure and cross-bred cattle bred for beef, fat accumulates so as to produce an eminence on the rump on either side of the tail. Such accumulations are particularly marked in old cows which have been repeatedly fed up for exhibition.

In certain Bats (*Rhinopoma microphyllum* and *Taphozous nudiventris*) considerable collections of fat have been found surrounding the root

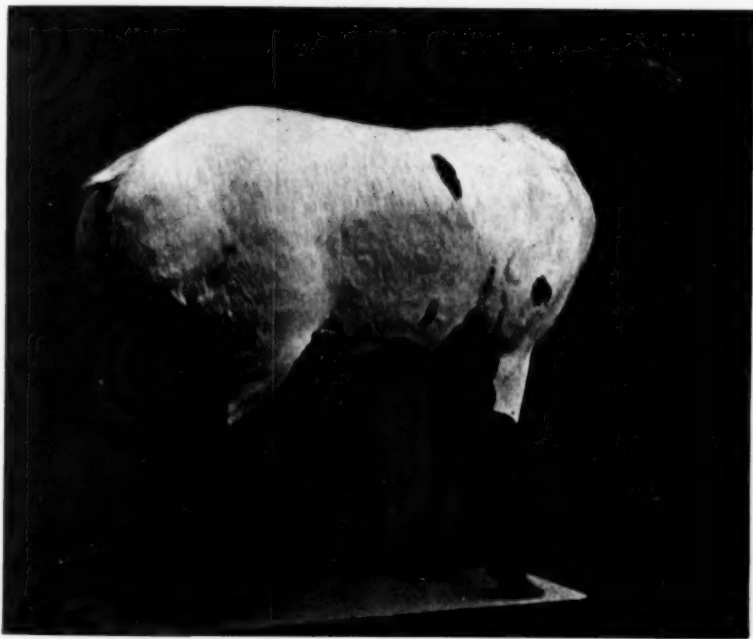


FIG. 12.

A fat-rumped sheep (*Ovis montanus*) from Hedjaz, Arabia. The tail is rudimentary. Photographed from the specimen preserved in the British Museum (Natural History), South Kensington. In this Arabian breed the head is black, the body and legs white; the lambs have a woolly coat; the coat in the adult is hairy.

of the tail and extending between the thighs, especially in specimens taken during the hibernating season.¹ In the specimen of *Rhinopoma*

¹ British Museum Catalogue, "Chiroptera," G. E. Dobson, pp. 387, 402.

microphyllum (flap-nosed bat) in the Museum of the Royal College of Surgeons there is a somewhat discoidal accumulation of fat beneath the skin of the abdomen, which extends downwards a short way over the upper part of the thigh, and beyond the genitalia and anus into the cutaneous fold, or interfemoral membrane, as far as the free portion of the tail. Microscopically this fat, I find, is of the common unilocular-celled kind. The absence of any marked accumulation at the base of the tail in this specimen must, presumably, be attributed to its having been taken at the close of the hibernating season.

But localized accumulations of fat in the tail may be deeply seated or intermuscular, and produce no evident external enlargement. In the tail of a large Monitor (*Varanus salvator*) Mr. William Pearson found during the course of a dissection, at the Royal College of Surgeons, two flattened symmetrical masses of fat many inches in length, deeply placed between the muscles. In this particular specimen they terminate 6 cm. from the base of the tail, and cease short of its distal extremity. They lie on the ventral aspect and on the outer surface of each of the two chief muscles situated on either side of the mid-line. Like these muscles, the fat does not come into relation with the skin; a zone of minor muscles intervenes. There are two smaller symmetrical areas of fat side by side, on the neural arches, on the deep aspect of the two muscles situated between the bones and the skin, which latter the muscles reach. The fat is quite unconnected with the copulatory sacs, which in the lizard lie, when in the retracted position, under the skin at the root of the tail, and which are represented by smaller though similar organs in the female. Histologically, the fat is of the common kind. The masses of fat are as sharply circumscribed and as isolable as any lipomata (which may be equally symmetrical); and their presence is the more striking since there is no fat in general between the muscles of the limbs or in the subcutaneous tissue. In the omentum there was a large discoidal mass of pure yellow fat. The only subcutaneous fat met with in lizards is that on the ventral aspect of the pelvis, whence it extends along the under side of the thigh; this has no relationship with the subperitoneal fat,¹ and is altogether so insignificant in amount, when present, that it may be neglected; its presence seems to be related to that of the "femoral gland." In a lateral dissection made to study the anterior or proximal termination of one of the chief pair of the intermuscular fat masses in

¹ G. W. Butler, *Proc. Zool. Soc. Lond.*, 1889.

the tail of the monitor, the fat is seen to be divided into coarse lobules, of discoidal form from lateral pressure; a single lobule of this kind constitutes the actual termination of the long adipose process. When unravelled the mass is resolved into groups of discontinuous lobules devoid of any proper longitudinal disposition.

In examining the tails of different lizards for intermuscular fat, in some I found none (as in the great American monitor, *Teius teguexim*); in others, a varying number of intercalated processes. In *Varanus griseus*, a female, the cross-section of the tail, 2 in. from the cloaca, showed two symmetrical areas of fat on the ventral aspect, having the same relations to muscle as in *Varanus salvator*. The most marked



FIG. 19.

A horizontal section of the tail of a Lizard (*Varanus salvator*) showing two symmetrical areas of pure fat deeply seated on the ventral side, between the muscles. On the dorsal aspect, on either side of the mid-line, there is a smaller area of fat which lies beneath the main muscle intervening between the skin and the arches of the vertebrae. (Museum of the Royal College of Surgeons.) Natural size.

example, however, was furnished by a male specimen of *Tupinambis nigropunctatus*. The cross-section, made 3 in. from the cloaca, displays two fatty processes lying ventralwards, two lying dorsally against the neural spine, and two laterally, as represented in the accompanying figure. The several processes terminate short of the skin and the superficial series of muscle columns. The fat is histologically of the common kind, and without any traces of included muscle fibre. Between the fat cells lie, here and there, highly branched, grotesque masses of

pigment, doubtless contained in cells. No subcutaneous fat was present in any part of the animal, and none between the muscles in cross-sections of the limbs. The different processes of fat I traced forwards by separating and removing the muscles; each terminates in the same manner, at varying distances from the cloaca. The ventral process, for instance, reaches to within 2 cm. of the latter; the lobules composing it become more and more laterally compressed and attenuated, and form, at length, a discontinuous longitudinal series. The paired copulatory cæca lie to the inner side of the two ventral processes for part of the length of the latter. That the fat is not a subperitoneal extension in connexion with the cæca which lie between the caudal muscles is shown by its ceasing short of the abdomen, and by the fact that the dorsal processes of fat terminate in the same way, about 2 in. from the

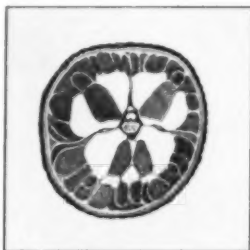


FIG. 14.

A horizontal section of the tail of a Lizard (*Tupinambis nigropunctatus*), showing a symmetrical mosaic produced by the divided muscles and sharply defined areas of fat intercalated between them. The section is made 3 in. from the cloaca. (Museum of the Royal College of Surgeons.) Natural size.

level of the cloacal aperture. In a second example of *Tupinambis nigropunctatus* the cross-section of the tail, 2 in. from the cloaca, exhibited two ventral processes of fat, one on the outer aspect of each of the main ventral muscles, and ceasing short of the skin. Histologically, the fat was without intermixture of muscle fibre, and was arranged in loosely applied and laterally flattened lobules. This shows that in the same species the fat is not of necessity similarly represented. In the short, vertically flattened tail of *Trachysaurus rugosus* I found a ventral and a dorsal pair of fatty processes intercalated amongst the muscles. The distribution of the fat was strikingly brought out by placing a thin slice for 24 hours in 80 per cent. alcoholic solution of

Sudan III. The substance of the different muscles was coloured pink, the fat was of a vivid crimson, and thin lines of it extended in the septa between certain of the muscles, beyond the limits of the main processes. Microscopic sections showed that the fat was confined to the inter-muscular connective tissue.

The presence of fat in the tail of certain of the limbless *Amphisbænidæ* was noticed by Butler in 1888, and before that had been described by Carl Smalian.¹

The fat in the cross-section of these different lizards' tails forms, with the muscles, so symmetrical a pattern that the question arises whether it does not represent muscles which have been replaced by adipose tissue. Such a substitution I have traced in the tail of the *Potoroos platyops* (one of the sub-family *Hypsiphrymninæ*, or kangaroo rats). The tail, which I had the opportunity of examining, through the

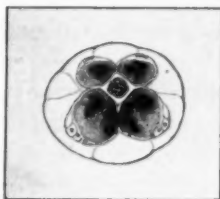


FIG. 15.

A horizontal section of the tail of *Potoroos platyops*, a kangaroo rat, showing the substitution of fat for disappearing muscle. The areas of muscular tissue still remaining are shown of a much darker colour than the fat; the small peripheral points are slender tendons. The form of the muscles is unaltered; they are surrounded with subcutaneous fat (left untinted). The body of a vertebra lies in the centre. (Museum of the Royal College of Surgeons.) Natural size.

kindness of Mr. R. I. Pocock, measures 9 in. in length and is cylindrical in form, though for the terminal $3\frac{1}{2}$ in. it is slightly swollen from a subcutaneous accumulation of fat, and measures here $\frac{9}{10}$ in., as against $\frac{6}{10}$ in. where narrowest. Transverse sections of the tail show that the muscles have, for the most part, been replaced by fat, without undergoing any change in form or size. Ill-defined areas of muscular tissue are recognizable in the midst of the four sharply defined masses of fat which represent the muscles. The microscopic sections demonstrate

¹ *Zeit. f. wiss. Zool.*, 1885, xlii.

very beautifully that the muscular substance is disappearing by a process of simple atrophy; the muscle fibres are in all grades of diminution in their sectional area, but retain a perfectly normal structure, whilst between them there is an abnormal growth of common adipose tissue. The process is one of simple atrophy, accompanied with the substitution of adipose tissue—*liposis*, as the condition should be termed, rather than *lipomatosis*, which would imply the growth of a tumour-like production of fat and should be reserved for such. *Mutatis mutandis*, liposis is the counterpart of fibrosis; lipomatosis, of fibromatosis.

And as showing the substitution completed, I may cite the specimen in the College Museum (No. 577 C), which is a transverse slice from the tail of an ox, in which the several muscles have been quite replaced by adipose tissue. The form of the muscles around the caudal vertebrae can still be discerned by the septa of connective tissue enclosing them, but they are, without being reduced or increased in size, completely replaced by fat. The cause of this replacement was not investigated.

To return to the fat in the tails of lizards, although their symmetry and size suggest an origin in vanished muscle, such a view I have not been able to verify. In none of the fatty areas microscopically examined have I seen any trace of muscle fibre—the adjacent muscles are normal, the transitional links are wanting, and lastly, as traced by lateral dissection, the fat is disposed in isolable flattened lobules unsuggestive of such a source. We may therefore regard these accumulations of fat in the tails of lizards as proper stores, although they are as isolable as "circumscribed" lipomata, and occur in a position where fat would not be expected, seeing that it is as absent from the general intermuscular as it is from the subcutaneous tissue.

This substitution of fat for disappearing muscle opens up the question how far anything similar obtains as an evolutionary phenomenon in the human subject, as distinguished, that is, from the fatty substitution occurring in the case of muscles that have been paralyzed or forced into inactivity. The disappearance of particular muscles in the process of evolution, and their replacement by fibrous tissue, is a well-established fact; but whether any tracks of fat represent muscles which have vanished during evolution is a subject worth inquiry, although the fact itself would be very hard to demonstrate. The fatty replacement of disappearing muscle in the individual, as distinguished from the species, presents less difficulty. The extensor and the curvator coccygis are just such muscles as we might anticipate to exhibit the substitution.

In man there is occasionally found, arising from the back of the sacrum, an extensor, and from the front a curvator of the coccyx, which represent the corresponding caudal muscles in the lower animals. Some dissections of my own show how largely fat may be intermixed with, and eventually replace, the muscular substance of the curvator coccygis. In a man, aged 25, who died of acute intestinal obstruction, the muscle, which was paired, consisted on each side of a band 0.7 cm. in breadth and 3.5 cm. in length, passing from the terminal segment of the sacrum to the side of the coccyx. The fleshy fibres in it were obvious by their colour, but they were largely intermingled with adipose tissue, there being as much fat as muscle, and a microscopic examination showed the intervention of extensive groups of fat cells, some in longitudinal rows, between the muscle fibres. In another dissection, the parts being obtained from a woman, aged 34, who died of gastric ulcer, there was in the same position, and covered with a thin fascia, a flattened process of fat, the sagittal sections of which showed a certain number of bundles of striated muscle fibre in its midst. In a third dissection, from a woman, aged 35, who died of cirrhosis of the liver, I found, on dissecting off a thin layer of fascia from over the site of the curvator, only a flattened collection of fat, the microscopic examination of which revealed no muscle fibre whatever.

Darwin¹ cites the opinion of Mr. Hodgson (*Journal of the Asiatic Society of Bengal*, xvi, pp. 1007-16) that the caudal enlargement of fat-tailed sheep in most of its phases is an instance of degeneracy. If by this it is meant that there is an histological deterioration of tissue, the view is incorrect. Hunter's preparation clearly proves that this is not so in the broad-tailed variety, for the muscle around the caudal vertebræ is compact and of normal colour, the accumulation of fat being wholly subcutaneous.

Of the accumulations or stores of fat in the subperitoneal and retroperitoneal tissue (around the kidney, for example), in the mesentery, and omentum, these are so general, not only in mammals but in *Sauropsida*, that they have no special interest. The so-called "fat masses" of lizards and snakes come into this category. In snakes they extend from the cloaca to the hinder margin of the liver, and form lobulated masses projecting into the abdomen which may be many feet in length; they are well developed whilst in the egg.²

¹ "Variation in Animals and Plants under Domestication," 1st ed., 1868, i, p. 93. (This view is repeated in the author's second edition of 1875.)

² Butler, loc. cit.

The Adipose Fin in Salmonidæ and Cetacea.

In fish the most widely known example of local fat-accumulation, although it is insignificant in size and can scarcely be called tumour-like, is furnished by the *Salmonidæ* and certain of the *Siluridæ*. In the salmon and some species of *Siluridæ* (between the families *Esocidæ*, pikes, and *Salmonidæ*) the second dorsal fin is represented by a lamina of fat—the adipose fin, as it is named. In a specimen of *Synodontes serratus* (one of the latter group) at the College of Surgeons, measuring 26 cm. from the mouth to the fork of the tail, the eminence in question is 7 cm. long, 2 cm. high, and 1 cm. where thickest at its basal attachment; its outline is somewhat hemi-elliptical, its free edge thin and fin-like.

Microscopic sections made of this fin, parallel with its flat surface, show that it consists solely of fat of the common unilocular-celled kind, supported by a somewhat open mesh of connective tissue. In the salmon the adipose fin is relatively much smaller. In a fish of the common size, *e.g.*, about 3 ft., it would not measure more than 3.5 cm. in extreme height (taken in the long axis), 1.5 cm. sagittally, and 0.5 cm. in thickness. It appears as a pliable, somewhat fleshy, leaf-like process, which comes cleanly away with the skin when the latter is peeled off from the muscular substance. A horizontal section shows it to consist mainly of intersecting bundles of stiff-looking, coarse fibrous tissue; along its middle is a narrow, ill-defined zone of fat, the cells of which are broken up into groups by intruding bundles of the general fibre; a few vessels, but no further structures, lie in the section. In these cases the eminence must be viewed as a growth of fat which has taken place in the residues of connective tissue representing a vanished fin.

The dorsal fin present in most of the *Delphinidæ* (dolphins and porpoises) is of a somewhat similar nature, in that it contains no bone, but is composed of tissue, partly fatty and partly fibrous. There is such a fin from the porpoise in the College Museum, which I have examined microscopically; it is invested with a thick, pigmented epidermis, into the deeper half of which there project narrow, close-set parallel cores, or papillæ, of connective tissue. The fin consists of intersecting strands of dense, fibrous tissue, in the meshes of which lie fat-cells of the common type. Beneath the papillary processes the bundles of fibre are, for a short distance, disposed parallel with the free surface, and the intervals between them occupied by fat-cells. A similar fin occurs, also, in many

other *Cetacea*—in the fin-backed whalebone whale, and in the toothed, bottlehead, and sperm whales.

As a physiological reserve the adipose fin of the salmon is, of course, altogether insignificant. In the salmon the general muscular substance is the great storehouse of fat, and it is upon this that the fish lives after it leaves the sea and ascends the rivers to spawn, for during the whole of this period, amounting often to several months, it takes no food from without. "The marked and peculiar degenerative changes which the

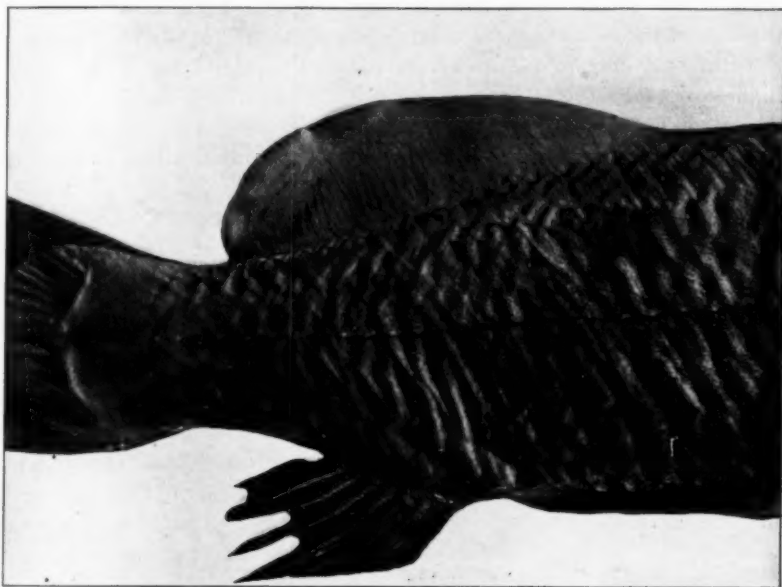


FIG. 16.

Showing the adipose lamina representing the second dorsal fin in the Siluridan *Symodontes serratus*, referred to in the text. (Museum of the Royal College of Surgeons.) Natural size.

lining membrane of the stomach and intestines undergo during the stay of the fish in fresh water shows that during this period the organs of digestion are functionless."¹ In the salmon leaving the sea for spawning

¹ Fishery Board of Scotland Report of Investigations on the Life History of Salmon, 1898, p. 171.

the fat is found in large amount in the connective tissue between the muscle fibres, as well as in the muscle fibres themselves. In the late fish of the upper reaches of the rivers ascended, the fat is found to have disappeared, having been partly transmitted to the sexual glands and partly used as a source of muscular and other energy. Although fat lies within the sarcolemma, the phenomenon cannot rightly be named degenerative, as is pointed out in the report referred to. The essential conception of fatty degeneration is *damage* sustained by the cell or muscle fibre, whereby the fat reaching it, or contained in an occult form within it, becomes evident from its being unutilized. In the case of the salmon, there is no reason whatever for thinking that the fatty muscle fibre is damaged; the muscles, on the contrary, are in the highest degree of efficiency, as proved by the marvellous manner in which the fish will overcome obstacles to its ascent. One must class the phenomenon as one of fatty infiltration of the muscle fibre and see its parallel in fatty infiltration of the hepatic cells.

The occurrence of true fatty tumours in fish is rare. In the College Museum there is (Specimen No. 332 B) the anterior pectoral fin of a roach, with a portion of the body-wall; beneath the skin at the base of the fin is an oval tumour, slightly over 1 in. in diameter, which is histologically composed of pure fat.

Wherever it is present, in health, fat should be viewed as food reserve, whether in the subcutaneous or intermuscular tissue, within the bones, or the cells of cartilage, or of glandular organs. In its wide distribution the fat in animal tissues is comparable to that of starch in the vegetable kingdom, though the glycogen of liver and muscle, chemically, of course, bear the closer comparison. Yet fat and starch are physiologically interchangeable.

The abundance of starch in leaf parenchyma, which is the great site of its production, and its abundance, *e.g.*, in the stem and branches of exogens, where it is stored in the cortical parenchyma, medullary rays, the woody parenchyma, and young medulla, are familiar biological facts. From these various positions the material is periodically being removed for use, and as constantly replaced. When a starch-containing tissue undergoes a natural process of death, the starch is removed from it without being replaced. The leaf that has just fallen in autumn is starchless, and I have found the same to be true of the dead portions of woody branches whilst they are still undetached—none of the cells any longer hold starch.

And, as if to complete the likeness, we have amongst plants tumour-like formations of reserve—pseudo-amylomata they might be named. The tuber of the Potato is the most familiar example. Similar starchy swellings constitute the corm of the Crocus, Colchicum, &c.

In the tropical *Dioscorea sativa* (Otaheite potato) starchy tubers grow in the axils of the leaves; they are furnished with buds, or "eyes," from which the plant is reproduced, upon this modified form of branch being shed; in the tropics they are well-known objects of sea-drift, in which they germinate. As an interesting abnormality, the growth of tubers above ground, in the axils of the leaves, has been noticed in the common potato.

The green pseudo-bulbs of Orchids are further similar examples, although the amount of starch reserved in these is not particularly abundant.

Other tumour-like swellings of reserve in plants consist of inulin, as is the case in the tubers of *Helianthus* (Jerusalem artichoke) or the fleshy roots of the Dahlia.

The removal of this substance is brought about by a special enzyme—inulase. The utilization of the fat reserve in many oily seeds is effected by means of lipase. The actual method of removal of both starch and fat, *mutatis mutandis*, is the same. Diastase is almost universally distributed through the several tissues of the plant, and effects the removal of starch for utilization elsewhere, or brings about its transport to a distant part, in which it is reconstructed and again deposited. In this way it is periodically removed from the leaves during darkness, the enzyme being formed in markedly less amount in sunshine. Besides this diastase of transport, or translocation, as it has been named, there occurs, from the scutellum of the embryo in the germinating seed, a more pronounced local secretion of the ferment ("diastase of secretion"), which acts upon the adjoining endosperm.¹ In the animal body, somewhat in the same way, the lipase which brings about the utilization of fat is found, not only in the pancreatic secretion which is shed into the intestine to ferment fat ingested (and, in a way, answers to the "diastase of secretion"), but, like diastase of translocation, lipase is widely distributed through the organism—in the liver and in the testicle in particular amount, as well as in the blood-plasma. As diastase is present in starch-holding tissue, so also is lipase in adipose tissue. The incitement to fat removal is the demand of the distant

¹ J. Reynolds Green, "The Soluble Ferments and Fermentation."

tissues, the whole phenomenon being one of chemical co-ordination. There is, perhaps, an increased output of lipase from the starving vital organs, which reaches the fat and hastens its removal. It has been proved that starvation is the stimulus which incites the secretion of diastase by the embryo: diastase does not appear in germinating barley until the fourth day, when the embryo, having utilized all its own nutriment, is forced to draw upon that of the endosperm. As an alternative, it might be assumed that the action of the lipase in adipose tissue is kept in check by an antilipase furnished by the other organs, and that the supply of fat to the starving tissues is brought about by a diminished output of the antibody. Some method of co-ordination, however, between the general wants and the draft upon the reserve supply must be assumed, in order to explain the results actually witnessed.

The Fat Masses in the Necks of Cretins.

Owen¹ cites Simon to the effect that in the Marmot, extending from the root of the neck to the posterior mediastinum and along the cervical vessels to near the mandible, there are bodies consisting of fat which undergo periodical increase in autumn prior to hibernation. The same author refers again to the observations of Handfield Jones, that in the hedgehog there are two roundish masses in the neck and others in the axilla "which might be modifications of adipose tissue." A dissection of the masses in the hedgehog has been lately added to the College Museum. They are symmetrical and of considerable dimensions, for flattened extensions lie on the deep aspect of the scapula, whilst others reach upwards towards the head beneath the trapezius muscle; the chief portion occupies the supraclavicular fossa, whence processes pass between the pectoralis major and minor to the axilla, and superiorly as high as the submaxillary salivary gland. These hibernating glands, as they have been named, are distinguishable, on dissection, from the common fat by their firmer texture and reddish colour. During hibernation these reserves are used up and dwindle away, a residuum of connective tissue alone remaining, to be again replaced before the arrival of the next hibernating season.

Similar "glands" occupying a like position are present in the dormouse (*Muscardinus avellanarius*). In the shrewmouse there are

¹ "Anatomy of Vertebrates," 1868, iii.

hibernating glands situated on the shoulders, and consisting of four symmetrical tongue-shaped masses of yellowish tissue each about 16 mm. long and 8 mm. at its broadest part. These enlarge as winter approaches and disappear in spring.

In the Hedgehog, for the first month of hibernation the weight of the "glands" falls rapidly, and the general fat is also removed; subsequently only little fat is removed from the gland until the end of March, by which time all the fat stored in the tissues is exhausted. From April onwards the gland is the only source of supply, and is eventually reduced to a mere fibrous cord by the time the animal awakens into activity.¹ The histology of the "gland," during October, in the hedgehog has been investigated by Carlier.² It consists of lobules each surrounded by a fibrous capsule which often contains much adipose tissue. Occasionally small lymphatic masses are present both in the septa and the capsule. The lobules consist of polyhedral cells, each enclosing a protoplasm with a wide-meshed network, in the interstices of which various-sized droplets of fat may be seen. Sometimes, though rarely, a single large drop of fat lies in the cell, but a certain amount of protoplasm remains between the fat and the exterior, and the nucleus retains its spherical shape, or may be slightly flattened. The nucleus usually occupies the centre of the cell with the fat droplets around it, leaving the periphery of the protoplasm almost free from fatty matter.

From a microscopical examination of the gland of the hedgehog, in an animal killed by means of hydrocyanic-acid vapour, October 13, 1908, I can confirm Carlier's account, except that the amount of fat in the cells is considerably greater than he has figured, a difference probably due to his not having used such an efficient fat dye as Sudan III (introduced by Daddi in 1897), for he examined the gland in the same month of the year—viz., October. The gland is sharply circumscribed, the fat around it being of the common kind with unilocular cells, *i.e.*, the cells around are distended with a single droplet of fat.

The cells of the gland itself, whether of the cervical or axillary portion, are of large size and the cytoplasm multilocular; some of the vacuolation is as fine as that of the cells of a sebaceous gland, but it is not so uniform, and spaces of larger and varying size occur amidst the more finely vacuolated substance of the cell; sometimes there is a

¹ E. W. Carlier, *Journ. of Anat. and Phys.*, Lond., 1892-3, xxvii, p. 509.

² *Ibid.*, 1892-3, xxvii, p. 508.

central drop of moderate size so situated. The character is strikingly brought out by the use of Sudan III, which stains the individual droplets of a brilliant orange-red. The cells lie in the meshes of a very close capillary network, the high vascularity being doubtless the chief cause of the difference between the colour of the tissue and that of the general fat. I encountered no lymphatic or hæmolymp tissue in the areas examined. This difference in the cell vacuolation is the more striking when it is remembered that, the animal being fully grown, the question of its representing a foetal or developing phase of fat does not arise. An examination of the general subcutaneous fat shows it to present nothing unusual; it is of the common unilocular-celled kind. I may note, however, that, although in this character the gland-tissue is well differentiated, in certain peripheral lobules presenting the typical structure, a fair number of unilocular cells of the common kind are intermingled; and certain of the peripheral lobules merge into those of the common fat. One is reminded of the transition between parathyroid and thyroid tissue as it may be seen in the parathyroid gland, which has led some to regard the former as simply an embryonic or incompletely developed form of the second. The rich blood-supply of the hibernating gland affords an explanation of the readiness with which fat may be either stored in or removed from the cells as occasion requires.

Mr. A. Mavrogordato was good enough to submit samples of the gland and of the common subcutaneous fat to analysis, in order to ascertain if there was any difference in the iodine value of the fatty acids. Hedgehog (1), killed with hydrocyanic-acid vapour, October 13, 1908: This method was resorted to in order to obviate the use of chloroform or ether, which might act upon the fat. The hibernating glands were at once dissected out; they were of a dull brownish colour, well defined, and more compact in consistence than the general fat. The subcutaneous fat was fairly abundant, pale yellow, and soft. There was no marked accumulation of fat in the omentum, large areas of the membrane being fatless. The amount of fatty acids obtained from the gland was less, proportionally to the bulk of material used, than in the case of the subcutaneous fat. The acids of the subcutaneous fat had a remarkably high iodine value; the iodine value of the general fat varies in different animals, but that of the hedgehog exceeds any of them. The iodine value of this was 90. The iodine value of the acids from the hibernating gland was 100. This difference is too small to have any significance. In the case of man, the common connective-tissue fat has an iodine value of from 60 to 70; the fat from the liver has a value of

from 110 to 130. Such a difference is very striking. Hedgehog (2), killed with hydrocyanic-acid vapour, October 22, 1908: The results confirm the preceding. The iodine value of the acids from the subcutaneous fat was 92.5; the iodine value of the fatty acids from the hibernating gland was 96.3. As might have been anticipated, the iodine value of the fatty acids obtained from the liver of the hedgehog follows the general rule of being notably higher than that of the common connective tissue, as will appear from the following: Hedgehog (3), killed with hydrocyanic-acid vapour, December 5, 1908: To test the fat extractable from the liver, which was sliced and washed immediately after death, two samples were used. The iodine value of the fatty acids extracted from one was 125.4. That of the fatty acids extracted from the other was 129.9. The slight difference has no significance. Of the second hedgehog I examined the thyroid microscopically: it presented a perfectly normal picture; the vesicles were lined with a single layer of cells; the colloid was homogeneous and deeply stained with eosin.

From a consideration of these facts the question arose to me, Do the fat masses in the neck and axilla of human cretins represent similar structures, and is there any corresponding structure in the normal human neck? Both these questions I am able to answer in the affirmative, and, although the demonstration in the case of the normal human neck has been already published by Hatai, my own discovery of the same fact was made quite independently. In regard to the fat masses in the human cretin, it may be observed, then, that they correspond in their anatomical position with the glands of the hedgehog; they are equally symmetrical, and they diminish with the emaciation of their possessor. These masses were first described by Mr. Curling, who also showed that they were fatty in character. Curling's two observations are reported under the title of "Two Cases of Disease of the Thyroid Body, and Symmetrical Swellings of Fat Tissue at the Sides of the Neck, connected with Defective Cerebral Development."¹

Case I.—A female child, aged 10, presenting the typical marks of cretinism. At the outer sides of the neck, externally to the sternomastoid muscles, there were two tolerably symmetrical swellings, which had a soft, doughy, inelastic feel. Similar swellings, but smaller and less defined, were observed in front of the axilla. The thyroid gland could not be perceived. Death occurred from erysipelas, accompanied

¹ T. B. Curling, F.R.S., *Med.-Chir. Trans.*, Lond., 1850, xxxiii, p. 303.

with glossitis and stomatitis, from which the patient died exhausted. At the autopsy the body was found much emaciated: *the swellings in the neck were much less in size than what they had been prior to her illness.* They were composed of fat and occupied the posterior triangle on either side of the neck, dipping downwards behind the clavicles and filling the axillæ. They could be traced extending slightly over the infraspinal muscles and the lowest angle of the scapula. They were not enveloped in a capsule, but consisted of fat of a loose, lobular structure, which seemed under the microscope to be made up of connective tissue and fat globules. There was not the slightest trace of a thyroid body.

Case II.—A female child, aged 6 months, characteristically cretinous. On the sides of the neck, beyond the sterno-mastoid muscles, were two soft, symmetrical swellings, having a doughy feel, and incompressible. They were of an oval shape, lying obliquely across the sides of the neck and extending from the edge of the trapezius to the middle of the clavicle. Death occurred shortly after admission. The swellings in the neck were found to consist of superficial collections of fat tissue without any investing envelope, and loosely connected to the surrounding parts. No trace of thyroid could be discovered. Curling sagaciously writes: "It is highly probable that this abnormal secretion of fat was dependent on the absence of those changes which result from the action of the thyroid, or on some imperfection in the assimilating processes, consequent on the want of the gland."

These observations were repeated from the clinical side by Hilton Fagge in 1871.¹ In a paper on "Sporadic Cretinism occurring in England," that author records four examples of cretinous children, and in each of these supraclavicular swellings were present. And still later, in the "Report on Myxœdema,"² there is recorded the case of a cretinous female child, aged 12, in whom there was the characteristic "soft puffy swelling in each supraclavicular fossa."

From a dissection carried out upon a sporadically cretinous female foetus at term³ I have been able to verify the presence of these fat masses. There is, in this foetus, a marked excess of subcutaneous fat throughout the body; there is no excess around the kidney or in the subperitoneal tissue, mesentery, or omentum. The dissection of the right side of the neck, &c., displays a prominent mass of fat in the

¹ *Med.-Chir. Trans.*, Lond., 1871, liv, p. 155.

² *Trans. Clin. Soc. Lond.*, 1888, xxi (Supplement: Myxœdema Report, Pl. III).

³ Museum of the Royal College of Surgeons.

posterior triangle extending up the neck, beneath the sterno-mastoid muscle, on the outer side of the great vessels. In the axilla there is a well-defined mass beneath the pectoralis minor, above and below which it extends; it approximates closely beneath the clavicle to that in the neck, but is not continuous with it. The child was born in the Marylebone Workhouse in June, 1905, the mother being aged 20, and the labour lasting four hours. The mother had, at the time, a living child, aged 12 months, in perfect health.

What I venture, then, to suggest in regard to these fatty swellings in the necks of such cretins is that they are not mere fortuitous local accumulations of fat, but that they are enlargements of the structures found in the neck of the hedgehog. As Curling surmised, the accumulation of fat in cretinism (as in myxœdema) is to be associated with the defective metabolism due to the diminished output of thyroid secretion. Together with the other marks of these diseases, the abnormal



FIG. 17.

A cretinous child, aged 1½, showing the fatty swellings in the neck and axilla.
The hair was very scanty.

accumulation of fat likewise disappears under the use of thyroid extract, and so do the fat masses of the neck and axilla. This disappearance was recently observed under thyroid treatment in the case of a cretinous child, aged 1½, under the care of Dr. F. J. Poynton, at the University College Hospital, through whose kindness I am able to furnish the accompanying figure.

In a second typically cretinous foetus at term I was able, by means of a horizontal section carried through the base of the neck, to demonstrate the presence of the peculiar gland-like tissue occupying the same position as in the hedgehog. To the naked eye it is easily distinguishable by its regular lobulation, being strikingly like a section of the

pancreas, and by a lesser degree of transparency, and slight difference in colour from the general subcutaneous fat which was itself in excess; its specific gravity, moreover, is higher than that of the latter. The microscopic examination of the tissue, made by means of Sudan III, reveals the fact that the large polyhedral cells comprising the "gland" are not of the common unilocular kind, but densely loaded with multiple droplets, not so fine as those, *e.g.*, of a sebaceous gland, but of a size to give the cell a moruloid character. In contrast, the adjacent subcutaneous fat of the same foetus shows the common unilocular type of cell.

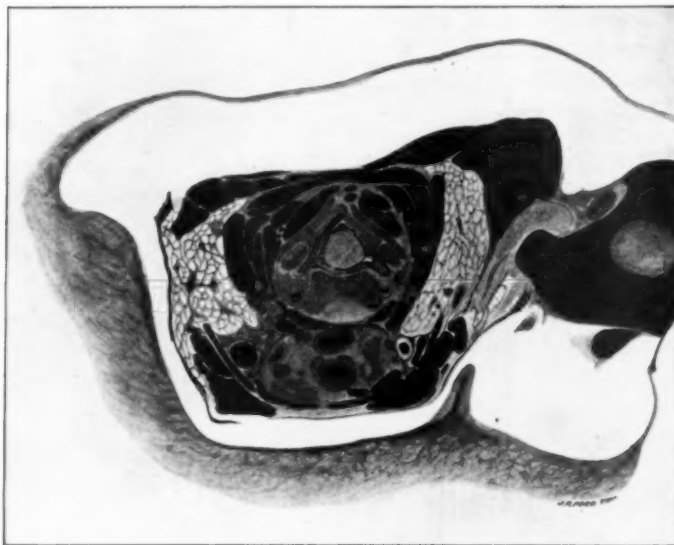


FIG. 18.

A horizontal section carried through the root of the neck of a sporadic human cretin at full term, showing the peculiar fat, the lobulation of which recalls that of the pancreas, occupying the position of the so-called hibernating gland of the hedgehog and other mammals. On the left side the section traverses the highest part of the scapula (the shoulder having been raised). Museum, Royal College of Surgeons. Natural size.

The histological question that here arises, whether there is any tissue in the anatomical position of a hibernating gland in the human neck which admits of differentiation from the common fat by the

characters already noticed, I am able to answer in the affirmative, although, as I have before stated, the publication of my own observations has been anticipated by those of Hatai. In a critical study of microscopical sections of the deep-seated fat of the neck made for this purpose I found that the tissue presented exactly the characters of the "gland." In a human foetus (of about five months), whilst the common subcutaneous fat showed the usual structure and consisted of unilocular cells of varying size, that about the great vessels presented a markedly different aspect. All the cells of this are multiloculated, with a well-stained net of cytoplasm, though not so finely vacuolated as are the

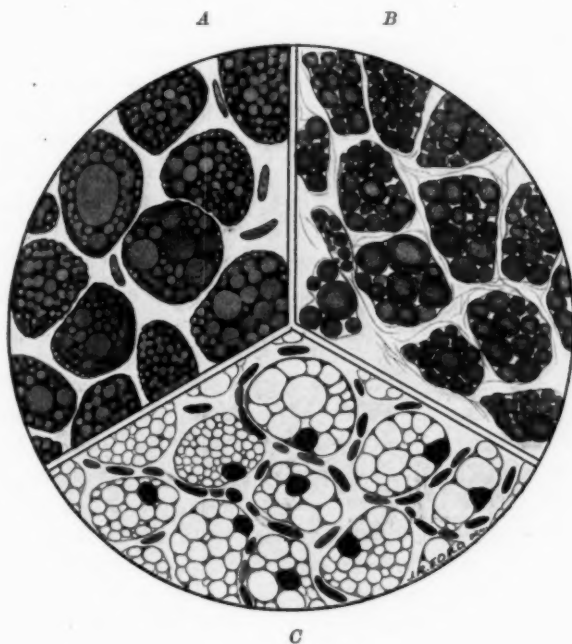


FIG. 19.

Showing the peculiar character of the fat of the "hibernating" or "inter-scapular gland" (of Hatai) from three different sources. *A*, from the cervical portion of the gland in the hedgehog, examined in the month of October; stained with Sudan III. The cells are loaded with fine droplets of fat. *B*, the corresponding tissue in the neck of a sporadic human cretin at full term; stained with Sudan III. The cells are moruloid from distension with droplets of fat. *C*, the corresponding tissue in the neck of a normal human foetus before term; hematoxylin and eosin; showing a similar moruloid condition. All the figures are drawn under one-sixth objective.

cells of a sebaceous gland. The fat droplets are of larger size in some cells than in others; only here and there does a unilocular cell occur amidst the others. The multilocular cells are arranged in distinct lobules separated by septa of connective tissue. Lying close to the carotid artery, and surrounded at the sides by this fat, are two lymphatic glands, and there is a small nodule of lymphatic tissue in its mid substance. E. Bonnot has recently re-described the gland in anatomical detail as it occurs in the human subject,¹ retaining the original name (though not a good one) given to it by Hatai—viz., the interscapular gland. Bonnot points out that the fat mass was described by F. Merkel,² and compared by him to the ball of fat in the cheek—the sucking-cushion, to which I will later refer. This comparison cannot for a moment be admitted. The fat of the latter is of the typical common kind and presents no histological peculiarity.

In the interscapular gland there is a variable number of lymph and hæmolymp nodes. These vary in different parts of the gland, and in some of its processes, that occupying the angle between the clavicle and scapula, and that extending horizontally from this towards the centre of the interscapular region, such nodules are wanting. Though called in the hedgehog the hibernating gland, and though actually serving as such, the fat in its cells being removed after that of the general connective tissue has been utilized, it is found, as Bonnot observes, in most, if not all, mammals. One may see it particularly easily, I may say, in the foetal rabbit by merely stripping off the skin; the cells, as studied with Sudan III, are of the multilocular type, whilst those of the thin layer of fat over the lower part of the thoracic wall and between the muscles have the common unilocular character.

Dr. Batty Shaw³ has discussed the question how far any form of adipose tissue can be regarded as specialised, or distinguishable from the common fat. He is inclined to hold, with Fleming, that all fat arises as a deposit in common connective-tissue cells, and that none is specialised in a glandular form. Kölliker, on the other hand, regards fat as produced from special cells, the resulting tissue being comparable to that of a gland. Dr. Shaw's observations were made chiefly upon the subpleural fat in infants of different ages, and in the foetus. It is inter-

¹ *Journ. of Anat. and Phys.*, 1908, xliii, p. 43.

² "Topographische Anatomie," 1899, ii.

³ *Journ. of Anat. and Phys.*, 1901, xxxvi, p. 1, "A Contribution to the Study of the Morphology of Adipose Tissue."

esting to note his statement that it is quite easy in the dissection of the axilla of a new-born child to separate fully-developed fat from fat of the other form; the latter looks more pink, appears firmer, and does not adhere to the scalpel. He examined perirenal and axillary fat in certain other cases.

The histological data forthcoming lead, I think, to the conclusion that there are two forms of adipose tissue. One of these arises from a deposition of fat in common connective-tissue cells, the fat, though deposited at first in droplets, eventually coalescing into a single sphere. The other is confined to particular areas, the fat being deposited in discrete droplets and remaining so, a form which histologically allies the cell-groups to those of a ductless gland. The fact that in the hedgehog this gland-like adipose tissue in the neck does not, during hibernation, behave like the common subcutaneous fat (being reserved whilst the latter is undergoing absorption) indicates a physiological as well as an histological specialisation. The development of such glandular fat, or fat glands, in the neck and axilla does not, of course, exclude the possibility of its formation elsewhere; and the histological structure in the subpleural and perirenal fat noticed by Dr. Shaw would have to be thus explained. One must, moreover, recognize the possibility of such fat being eventually replaced by adipose tissue of the common variety, formed from the interlobular connective tissue, by a process of fatty substitution of the kind witnessed, *e.g.*, in the case of the thymus. Dr. Shaw has found that the multilocular-celled variety is not present in the subpleural fat in later life, and the same is true of that which is present in the neck at the time of birth.

In cross-sections of the deep-seated fat by the great vessels towards the root of the neck, from a well-nourished woman, aged 28, who died of meningitis, and in whom the general subcutaneous tissue presented a well-developed but not excessive layer of fat, I found only a few patches of moruloid cells, the rest of the fat consisting of lobules of the common unilocular-celled kind. In the fat were included four discrete lymph nodules or minute lymphatic glands. In similar sections of the deep fat about the cervical glands and the internal jugular vein of a child aged 9, dying of acute rheumatism and pericarditis, I did not come across any fat of the moruloid variety. And, lastly, one must be prepared to allow that the cervical and axillary masses may be increased in volume by the formation of common fat from the interlobular connective tissue, in the same way that the size of the parathyroids, *e.g.*, may be, from the same cause.

The Sucking-Cushion.

Besides tumour-like accumulations of adipose tissue which act as food reserves, there is a further function served in one position in the human subject which is altogether of a different character, and where it is primarily mechanical. The position in question is the cheek, in which, in the new-born child, there is a spherical mass of fat which lies upon the buccinator muscle immediately in front of the masseter, and, though elsewhere surrounded by the subcutaneous fat, is quite isolable and distinct from it. Virchow, in his incomparable work "*Die Krankhaften Geschwülste*" (1863), has drawn attention to the fact that this body was described so long ago as 1741 by Heister, who regarded it, however, as a gland, and named it the "*Glandula molaris*." More fully, as Virchow notices, it was described in 1853 by Gehewe, in an inaugural thesis entitled "*De corpusculo quodam adiposo in hominum genis obvio*," Dorpat. The reason why the body in question finds a place in Virchow's work (*Lecture on Lipoma*) is that it may give rise to a lipoma in the cheek which simulates a parotid tumour.

Mr. Edmund Owen¹ has recorded an example of tumour of the sucking-cushion which he excised. After incising the mucous membrane of the mouth, a lobulated lipoma of considerable size at once protruded, and was withdrawn by gentle traction. The patient was a lady who presented a painless swelling of the left cheek; after a meal this was sometimes more conspicuous. After the removal of the lipoma the cheek on several occasions swelled as badly as ever; and later on a calculus was felt which shifted its position over the masseter. The calculus was removed by incision through the cheek; it was about the size of a date-stone and lay in the parotid duct. Owen suggested that the irritation caused by the calculus brought about an over-nutrition of the fatty pad and so determined its hypertrophy. The removal of the calculus alone, he remarks, would not have restored the symmetry of the cheek. This suggestion is a very probable one, for we find accumulations of fat around the kidney which has been destroyed by calculous pyelitis, about the breast which is the seat of carcinoma, in the synovial fringes and villi in chronic synovitis, and in other cases. In all these the increased vascularity arising from the disease is attended with an increased vascular supply to the adjacent tissue and so brings about a local storage of fat. (Gehewe was the first to point out that the

¹ *Lancet*, 1890, ii, p. 71.

purpose served by this ball of fat is to prevent the cheek from being drawn in between the jaws during the act of sucking. Bichat, at the beginning of the nineteenth century, had described it correctly as consisting of fat, but without perceiving its function: "Il y a presque toujours à cette époque, entre le buccinateur, le masséter et les tégumens, une espèce de boule graisseuse qui fait un corps isolé de la graisse environnante et qu'on extrait en totalité: elle contribue beaucoup à la saillie remarquable que les joues font à cette époque de la vie."

The article by Ranke¹ contains references to the work of all previous observers, and is well illustrated, although anatomically it adds nothing material to the previous descriptions. Ranke, in accordance with the function assigned to the fat mass, has named it the "Saugpolster," or sucking-cushion. The interesting physiological observation added by that author is that the fat of the cushion does not undergo absorption during emaciation. He commences his dissertation, indeed, by remarking that in children in whom wasting occurs during the first year of life, from diarrhoea, one often sees, when the greater part of the subcutaneous fat has disappeared in both cheeks, a small swelling in the neighbourhood of the Stenonian duct, about the size of a split hazel-nut; when pressed between the fingers from within and outside the mouth it feels soft, somewhat like a cyst.

The sucking-cushion persists in the adult, though in a less pronounced form; and it is attached by a pedicle to the canine fossa. It is most readily displayed by means of a horizontal section carried through the face between the upper and lower dental arches. So well defined is it that it readily allows of being enucleated from the proper fat of the cheek; and I venture to represent such a section, in which I have on one side enucleated the cushion, as adding pictorially, perhaps, the only thing that can be added to Ranke's figures. After such enucleation the bed in the surrounding fat is seen to be lined with a distinct layer of connective tissue. Were it a morbid production, we should name the mass a circumscribed lipoma. Gehewe (loc. cit.) found it clearly present in the foetus as early as the fourth month. This I can confirm: at this age it is represented by a minute collection of fat lobules, recognizable by means of a hand lens. And one can easily demonstrate it by means of a horizontal section at all intra-uterine ages afterwards. In the accompanying figure I have introduced such a

¹ Virchow's *Archiv f. path. Anat. u. Phys.*, Berl., 1884, xcvii, 527.

section taken from a fœtus below term. Microscopical examination of both of these specimens shows the adipose tissue of the cushion to be identical in minute structure with that of the surrounding fat of the cheek, from which it is separated by a thin layer of connective tissue. Ranke's injected specimen demonstrates the presence of large vessels which run in the capsule and penetrate the fat, the cells of which are surrounded with a rich capillary network, Gehewe's statement that the mass was supplied only with few and small vessels being erroneous.

The capillary vascularity, however, is not in excess of that of the fat lobules of the subcutaneous tissue of the fœtus at term, where a fine injection displays a close net of vessels from which hardly an individual cell is left out. That the circulation or blood-supply of the cushion is peculiar, nevertheless, appears from an observation I had the opportunity

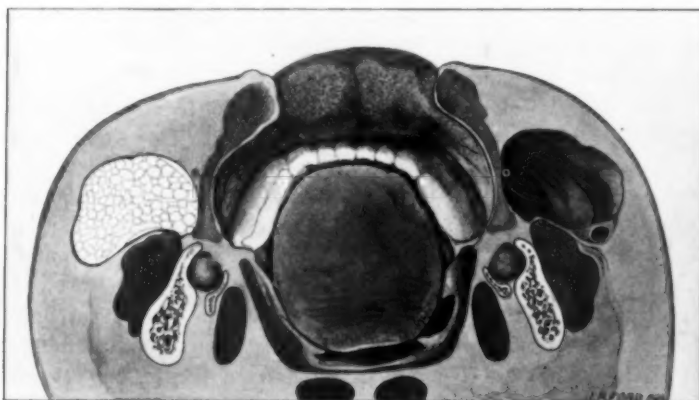


FIG. 20.

A horizontal section of the cheeks, including the rami of the lower jaw, of a sporadic cretin at full term, the lower of the two sections being represented. The knife was carried horizontally between the dental arches. The cretin was notably obese. In the sagittal sections of the labia majora a certain number of small fat lobules were recognizable. There was no abnormal amount of fat in the subperitoneal tissue. The sucking-cushion, shown on the left side, lying on the buccinator muscle and immediately in front of the masseter, is a well-defined ball of fat 2 cm. in extreme diameter. The lobulation of its fat is somewhat different from that of the subcutaneous fat, in the deeper part of which it lies. On the right side the cushion has been cleanly enucleated, the fossa in the general fat being lined with connective tissue. In the boundary of the lowest part of the fossa there is a thin sheet of muscle appertaining to the highest portion of the platysma. The upper half of the cushion was partially invested on the outer aspect by the zygomatici. The facial artery lies on the inner side of the cavity. (Natural size.)

of making upon a new-born child at term which had died from prolapse and strangulation of the umbilical cord. On cutting the cheeks horizontally between the dental arches, the two cushions stood out in a striking manner, being of a deep blackish-red colour from engorgement, whilst the fat around was quite pale; they were each 2 cm. in chief diameter, and so well encapsuled that their mere weight almost brought about their enucleation: and I have seen the same thing on a second occasion. The fat lobules comprising the cushion were differently disposed and more loosely held together, much, indeed, as in a salivary gland, which may well account for the body having been regarded by Heister as such. Ranke remarks (*loc. cit.*) that it would be of interest to determine whether the cushion is present or not in the anthropoid apes.

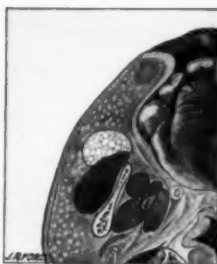


FIG. 21.

A horizontal section of the cheek carried between the dental arches, showing the sucking-cushion lying upon the buccinator muscle immediately in front of the masseter. From a human fetus below term. (Natural size.)

As examples of such I have been able to examine from the College stores the heads of two infant Chimpanzees (*Anthropopithecus troglodytes*). One of these, a female, had no teeth erupted; the umbilical cord was separated and the cicatrix formed. From the vertex to the tip of the coccyx the measurement was 10 in. In the horizontal section carried through the cheek between the jaws no sucking-cushion is displayed, nor is any fat discernible to the naked eye in the subcutaneous tissue of the cheek; and there was none in that of the buttock or front of the thigh. Nevertheless, a microscopic section of the parts revealed the presence of a group of fat cells in the position of the cushion and evidently its representative. In the second specimen the central incisors, both upper and lower, are just appearing through the gum.

As indicating its age, in the infant Orang reared by Wallace¹ the two upper incisors were cut at the age of five weeks, but the growth of the animal had been almost arrested owing to the want of its natural maternal nourishment. In the proper anatomical position there is a well-defined sucking-cushion 7 mm. in diameter. There is a thin layer of subcutaneous fat on its outer side, which extends for a short distance forwards in the cheek.

Passing lower down the scale, in a stillborn Sacred Baboon (*Papio hamadryas*) in which the umbilical cord was still attached, and the foetus at term, I found, on making the usual horizontal section, that the sucking-cushion was well differentiated, though not large. There was very little



FIG. 22.

A horizontal section of the cheek of an infant chimpanzee (*Anthropopithecus troglodytes*) showing the sucking-cushion in front of the masseter. (Natural size. Museum, Royal College of Surgeons.)

subcutaneous fat over the buccinator; the cushion is sharply encapsulated, spherical, not more than 6 mm. in diameter, and lies immediately in front of the anterior border of the masseter muscle.

Notwithstanding its presence, there is in both the Chimpanzee and Baboon a complete absence of the rotundity of the cheek which is so pronounced in the human foetus at birth, and which is indeed a peculiarly human feature. The absence of this rotundity is due partly to the small amount of fat there is in the subcutaneous tissue of the cheek in

¹ "Malay Archipelago," 1869, chap. iv.

general, and partly to the stretch or length of cheek itself arising from the extreme prognathism of the simian face, the sucking-cushions lying at the extreme posterior part of the cheek, and being, moreover, of relatively small size as compared with those in the human infant.

In a further new-born Guinea-baboon (*Cynocephalus sphinx*) with the cord still attached, the horizontal section through the cheek, made after the head was hardened, showed a minute group of fat lobules immediately in front of the masseter, in the regular position of the cushion, and evidently its appearing rudiment.

In the fœtus of a tailed monkey (the common bonnet) the horizontal section showed a very thin layer of subcutaneous fat over the masseter and a few minute lobules in front of it, but no definite cushion. In front of this there was no fat intervening between the buccinator and the skin of the cheek.

The sucking-cushion is not to be confused with the external eminence of subcutaneous fat present in the cheek of the River-hog (*Potamochoerus*). In this animal there is a prominent hemispherical swelling in front of and below the eye, the skin covering which is not horny or otherwise changed. In the East African river-hog (*Potamochoerus Johnstoni*) the swelling is particularly pronounced.

In a preparation (*Potamochoerus* ? species) in the College Museum (No. 1840A, Physiological Series) the eminence measures across its base 5 cm. by 3.5 cm., and vertically to the surface 1.7 cm. Its upper border is 5 cm. distant from the palpebral fissure. The vertical section shows it to consist solely of fat, continuous laterally with the comparatively thin layer beneath the adjacent skin, and closely connected with the skin over the eminence itself. Histologically, the fat is of the common kind, parted into lobules by processes of connective tissue continued into it from the suprajacent corium. The latter is not abnormally thick, and its epidermis presents no horny elevation. The structure of such facial protuberances, however, is not the same in the different species. There is in the College Museum the cheek of a wart-hog (*Phacocœrus ethiopicus*) showing, as in the living specimen in the gardens of the Zoological Society, two eminences on either side, one below the eye, and the other some distance in front of it. Both of these are composed of extremely dense fibrous tissue; the fat is limited to small groups of cells lying here and there, in insignificant numbers, in the midst of the intersecting fibre.

Why the fat of the sucking-cushion should withstand absorption during the emaciation of the rest of the body has not been satisfactorily

explained. Its persistence during emaciation serves the purpose of assisting the cheeks to functionate, and in this degree aids in the preservation or prolongation of life. Ranke suggests as a reason that it has a function to maintain till the last, and that it receives in consequence a richer blood-supply than the other fat of the body.

One may suppose that the mechanical function of bearing a higher degree of *intermittent* pressure than the surrounding fat leads to an increased vascularity, and, associated with this, one has to remember the isolation of the mass and the narrowness of its main attachment. Absorption would less readily occur from the vessels of such a pedicle than in the case of the general subcutaneous fat, the vascular connexions of which with the surrounding parts are more intimate. In one of my own observations upon a full-term foetus asphyxiated during birth, and to which I have before referred, the intense congestion of the two cushions, whilst the fat around was quite pale, indicates a difference in the circulatory mechanism, the return of the blood being less ready from the cushion than from elsewhere.

This persistence of fat in a particular spot, the sucking-cushion, in spite of its removal elsewhere, is matched in the muscular system. For whilst the muscles in general are utilized, and waste, during starvation, the diaphragm, the action of which is essential to life, remains well nourished and intact.

That the character of the fat in general is not constant, but may be modified by the kind of food ingested, has been long known amongst breeders of poultry and cattle. The fat of poultry, if the birds are fed largely on maize, is yellow, oily, and objectionable, instead of presenting the whiteness and firmness which result from feeding with oats or barley.¹ Such observations have been confirmed by the more accurate and scientific research of Leick and Winckler.² These observations showed that the composition of the fat in connective tissue varies, and that it is, moreover, not identical with that obtainable from the liver or heart muscle.³ The differences are determined by the variations in the iodine value of the fatty acids, *i.e.*, the percentage amount

¹ "Poultry," W. B. Tegetmeier, 3rd ed., 1898.

² *Arch. f. exper. Path. u. Pharmacol.*, Leips., 1902, xlviii, p. 163.

³ The fat in the tail of the fat-tailed sheep, it has been stated (by an observer cited by Geoffroy Saint-Hilaire and Frédéric Cuvier, *loc. cit.*), is more fluid than that elsewhere. This the Hunterian specimen at the College of Surgeons bears out; the fat is soft and of a pale yellow, as contrasted with the hard, white fat of ordinary mutton. So, too, Pennant ("History of Quadrupeds," 3rd ed., 1793) states that the fat tail is of a "substance between fat and marrow."

of iodine absorbed by the fatty acids after their isolation. In certain of the observations referred to the iodine value of the fatty acids works out as follows:—

						Subcutaneous fat
Normal dog	56.1
Dog poisoned with phosphorus	58.6
Normal sheep	38.2
Sheep poisoned with phosphorus	36.9
Dog fed with mutton fat after being starved, and subsequently poisoned with phosphorus	43.3

Although the precise method used in the extraction of the fatty acids in these experiments is open to question, this is mainly so when the method is applied to the viscera, and not when adopted for connective-tissue fat, the simplest from which the extraction can be made.

The possibility that the fat of the sucking-cushion owes its persistence to a difference of composition, so that it might conceivably be unaffected by a common circulating lipost, led me to put the case to Mr. A. Mavrogordato, to whom I handed over the material for investigation. He could detect no difference in the melting point, and none in the iodine value of the fatty acids extracted from the cushion and those from the common subcutaneous tissue.

Child at term; death from strangulation by the umbilical cord. The sucking-cushions were cleanly shelled out and placed in 10 per cent. salt formol; each had an extreme diameter of 2 cm. A sample of subcutaneous fat was removed from the shoulder and front of the thigh and similarly treated. Briefly stated, the method adopted was as follows: The fat is saponified by heating it with caustic potash, the whole of the tissue being thus dissolved; the fatty acids are then liberated by the addition of hydric sulphate; the liberated fatty acids are dissolved by shaking with petroleum ether; the clear supernatant fluid (petroleum ether) is removed with a pipette and submitted to distillation in a stream of carbon dioxide under reduced pressure, the fatty acids being left in the flask. The acids, after being weighed, are next dissolved in carbon tetrachloride and their iodine value estimated on the following principle: Acetic-acid solution of iodine of known value is added to the carbon tetrachloride; a certain amount of iodine is absorbed by the fatty acids. The amount is ascertained by adding solution of sodium thiosulphate until the whole of the colour is discharged and no further reaction is given to starch solution. The amount of standardised solution of sodium thiosulphate used is noted, and by this means the amount of iodine of which the colour has been discharged is calculated;

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on subtracting this amount of iodine from that originally added the amount absorbed by the fatty acids is arrived at, and, the weight of the acids being known, the percentage admits of being determined. Two samples of fat from the thigh and shoulder were used. The iodine value of the fatty acids proved to be—

53.2 and 52.5.

The fatty acids extracted from two samples of the sucking-cushion gave iodine values of—

57.6 and 58.1.

The differences brought out in the iodine value of the fat from the limb and that of the sucking-cushion are too slight to be of any significance.

I have, in conclusion, to express my thanks to Miss M. A. Murray, Mr. R. I. Pocock, Mr. R. Burne, and Dr. C. G. Seligmann for aid in furthering the collection of certain of the data brought together in this communication.

ADDENDUM.



FIG. 23.

The sitting figure from Knossos, Crete, referred to in the first part of the paper dealing with steatopygy, showing the typical Bushman form of the latter, unassociated with enlargement of the thigh. For this photograph I am indebted to Dr. D. G. Hogarth, the Keeper of the Ashmolean Museum, Oxford.

A Fatal Case of Streptotrichosis with Primary Lesion in the Lungs—the Organism Pathogenic for Animals.¹

By J. M. BERNSTEIN.

HISTORY.

A CARPET porter, aged 67, employed at a large store for twenty-seven years, was in fairly good health until five weeks before death, though his appetite had been failing for a fortnight previously. A sudden attack of pain of an aching character between the shoulders and at the lower part of the back caused him to take to his bed. In two or three days the abdomen began to swell, and the night before admission to hospital (two weeks after the onset) he coughed incessantly. He had wasted rapidly whilst in bed. Had had a slight cough, worse in the mornings, for four months. No history of pulmonary tuberculosis; bronchitis sixteen years before, with winter cough since; three years before had a bad illness with much wasting, but no night sweats, which lasted one month. Habits: Several pints of beer daily, with occasional exacerbations. Had been in the Army, but never abroad.

On admission the abdomen was distended, especially the upper part, with fluid; temperature, 98·6° F.; respiration, 24; pulse, 100; heart, *nil*; rhonchi and coarse râles over the lungs, posteriorly below the angle of the left scapula, a patch of dullness with blowing breathing. Urine *nil*. The temperature rose gradually with daily remissions during the first week to 101° F., and then to 102° F., and in the next week at times reached 103° F.; the respiration increased to 36, the pulse to 120, the cough became more troublesome, and he died nineteen days after admission and five weeks after the onset. A week before death 152 oz. of opalescent fluid were withdrawn from the peritoneal cavity. This was turbid, and unfortunately the routine examination only was performed, revealing leucocytes and epithelial cells; four days before death there were 12,400 leucocytes in the blood.

POST-MORTEM FINDINGS.

Body spare and wasted; some straw-coloured fluid in abdomen. Peritoneum everywhere covered with closely arranged minute granules

¹ From the Laboratories of the Westminster Hospital and the Brown Institution.

(size of ground rice), even in the pelvis. Abdominal viscera normal; spleen 8 oz., liver 64 oz., kidneys 6 oz. each. Pleuræ normal. Right lung 57 oz., in the lower lobe a mass of consolidation the size of an orange projecting slightly above the surface on the lateral aspect, where it appeared as an injected red area, mottled with white purulent foci. On section it was found to be honeycombed with small suppurating foci. Several smaller but similar areas were found in the same lobe, and a large area in the adjoining middle lobe together with several smaller areas and some minute spots of softening. Left lung, 16 oz., contained no such lesions. Both apices were shrunk and calcified, and everywhere throughout both lungs were small pigmented nodules due to chronic interstitial pneumonitis; mediastinal glands pigmented. Nothing further of note.



FIG. 1.
Section through lung.

The general appearances of the pulmonary lesions suggested actinomycosis, and a cursory examination of the pus revealed a Gram-positive and acid-fast streptothrix; consequently, various culture-media were inoculated with some of the contents of the deeper abscesses, and a further quantity was also sealed off in sterile pipettes for animal inoculation. This material from the cavities was thick, curdy, and white, and was drawn into the pipettes with difficulty. Next morning a guinea-pig and a rabbit were inoculated. Pure and prolific cultures were obtained in all the media and pathogenic effects produced in the animals. The details of these observations are given below:—

HISTOLOGY OF THE LESIONS.

Lung : In the consolidated areas are large collections of leucocytes, which have formed definite abscesses and increased in size by solution of the alveolar walls; the central parts are degenerating. The alveoli around these areas are filled with fibrin. In the less affected areas there is hyperæmia and the alveoli contain cells and fibrin, whilst further afield they contain some catarrhal cells in a patent lumen. In amongst the leucocytic infiltration numerous streptothrix filaments with branching portions are present.

Peritoneum and omentum : In these sections are found subperitoneal minute miliary tubercles with large central giant-cells, connective-tissue cells, &c., as found in the so-called typical histological tubercle due to *Bacillus tuberculosis*, from which it is impossible to distinguish the lesions. The giant-cells contain a great number of nuclei.

Sections stained with carbol-fuchsin and treated with acid and alcohol showed two or three deep-stained bacilli, an occasional one being found in a giant-cell; but, if anything, they were a little broader than *Bacillus tuberculosis*, though it must be recognized that it is impossible to differentiate between them. With Gram's method no filaments or any organisms could be found in the sections examined.

BACTERIAL FINDINGS.

Cultures were obtained on all the ordinary laboratory media under aerobic conditions only.

In *broth* a whitish surface growth had formed in twenty-four hours, and this in two weeks increased to a dense efflorescent growth, which adhered to the sides of the tube as a coherent membrane, for the most part thin and white, but in places finely nodular. The media remained clear, but portions of the surface growth had broken away and added to a slight layer of growth forming at the bottom of the tube.

In a flask of *bouillon* containing human blood, in a few days the surface was covered with a whitish membrane of growth.

On *potato* in a few days white, discrete, raised, hard colonies with a wavy margin and about the size of a pin's head, and in a few weeks this had increased to a white heaped-up mass of discrete colonies with irregular hard surface but with little tendency to spread widely. The surface was efflorescent.

On *agar-agar* a hard whitish growth slowly formed, resembling

actinomycosis, of discrete colonies, raised, hard and later heaped up, but never luxuriant.

On *glucose-agar*: a similar growth, but, early, in the condensation water there appeared discrete spheroidal white colonies which adhered to the sides of the tube on shaking.

In a *glucose-agar stab*: little growth in the depths but more towards the surface, over which it slowly spread with a wavy margin and heaped-up centre. In a *glucose-agar shake* there was no growth in the depths.

Blood-serum: as on agar, and no liquefaction.

Gelatin stab: a good growth spread over the surface, but diminished in amount along the track of the needle with lateral offshoots.

The characteristic surface growths on fluid media and the hard, discrete, raised growths on solid media were obtained in all subcultures, and also in cultures from the lesions in the experimental animals. On removing the surface growth from the bouillon flasks it showed no tendency to reform, growth proceeding in the depths whilst the media remained clear above.

All the cultures were strikingly white in colour, but an old potato growth has after some long time become faintly pinkish.

MORPHOLOGY OF THE STREPTOTHRIX.

The most notable feature about the organism is its pleomorphism. It is only seen as a branching filamentous growth forming a mycelium in the tissues and original pus and in very young cultures, whilst in artificial media it rapidly tends to split up into spheroidal, spore-like bodies and to grow as short rods. The filaments in the tissues and pus of the lungs and in the abdomen of the guinea-pig were not homogeneous on staining with Gram's method—the only satisfactory method we could use—but presented a beaded appearance with intervening faintly-staining areas.

In cultures this beading is seen early, and in the surface growth on bouillon, potato, &c., the threads rapidly split up into spheroidal "spore"-like bodies, so that in about a week the predominating feature is the collection of these spores between a few filaments. Often these filaments are very scanty and very faintly staining, and a Klatsch preparation from the surface of a few-weeks-old potato-culture consisted almost entirely of spores and resembled a film of staphylococci. In the deeper parts of the potato growth and in the deposit in the bouillon

tubes fine moniliform threads occur, probably degeneration forms; and in the latter numerous short rods resembling bacilli, and short beaded filaments, but never any spores, which only appear to form in the surface growths. In old cultures involution forms are found in the shape of filaments with small bulbous swellings in and at the extremities. In the guinea-pig's pus many collections of spores were found amongst the beaded threads. In later subcultures, made after some length of time, no mycelium was obtained, but short branching threads and short rods,

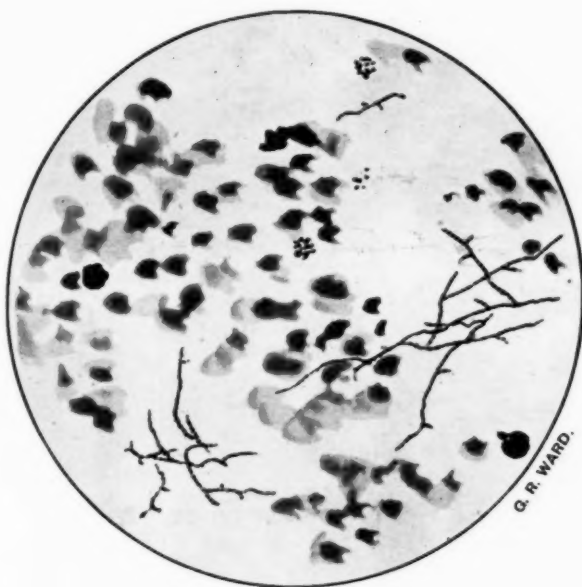


FIG. 2.

Streptothrix in pus, stained by Gram's method, showing beaded mycelium.

some beaded, formed the main portion of the growth, with but a few spores.

TINCTORIAL CHARACTERISTICS.

No satisfactory results could be obtained with Löffler's methylene blue, or with a modified Romanowsky, the latter producing only a granular appearance. Gram's method was most satisfactory. Ziehl-Neelson's method: In the original pus the filaments retained the stain

after treating with 25 per cent. sulphuric-acid methylene blue for five minutes or more, but even then they were not so deeply stained as tubercle bacilli. In the pus from the guinea-pig, after five minutes' treatment with this same stain, the spores remained red, but the filaments only partly so, and after one hour's treatment only the spores and some short rods remained red. In cultures the spores were more acid-fast than the rods or filaments, those in older specimens resisting the acid methylene blue for sometimes an hour.



FIG. 3.

Sixteen hours' culture in broth from original pus.

RESISTANCE OF THE ORGANISM.

The organism remained alive in sealed-up tubes of pus for quite three months and cultures were then obtained, but experimentally it was not noted as virulent, though perhaps this may be ascribed to the fact that only a little was left for inoculation. In old broth-cultures also the virulence seemed diminished, though larger doses did produce pathogenic effects.

Thermal death point: Cultures containing many of the spore-like bodies were unaffected by a temperature of 60° C. for thirty minutes, but were readily killed at 80° C. for ten minutes.

EXPERIMENTAL RESULTS.

Guinea-pigs.—(1) An adult male animal received intraperitoneally about 1 c.c. of the contents of a caseating area in the lung on the morn-

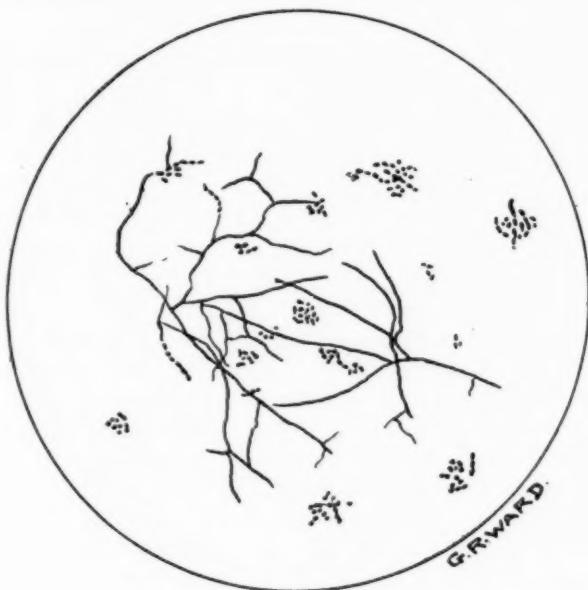


FIG. 4.

Seven days' culture in broth from original pus.

ing following the autopsy. The animal became acutely ill with a temperature of 105·6° F. next morning, rapidly wasted, and, as it appeared moribund, four days later was killed. A few miliary nodules were found on the peritoneum around the site of inoculation and some caseating nodules in the omentum about $\frac{3}{8}$ in. across. Pure cultures of the streptothrix corresponding to the original strain were obtained from the peritoneum and no other organisms. Histologically the miliary nodules

consisted of a subperitoneal cell-infiltration in which were some roundish areas made up of epithelioid cells—the arrangement recalling the miliary tubercle—but no giant-cells were found. In these and in the areas in the omentum the branching filaments described above were readily found.

(2) A second adult male guinea-pig received about 1 c.c. of a thick emulsion of the surface growth of a blood-bouillon flask three weeks later, and it also became acutely and rapidly ill, with a persistent temperature of 105° F.; in five days it was much emaciated and the testicles

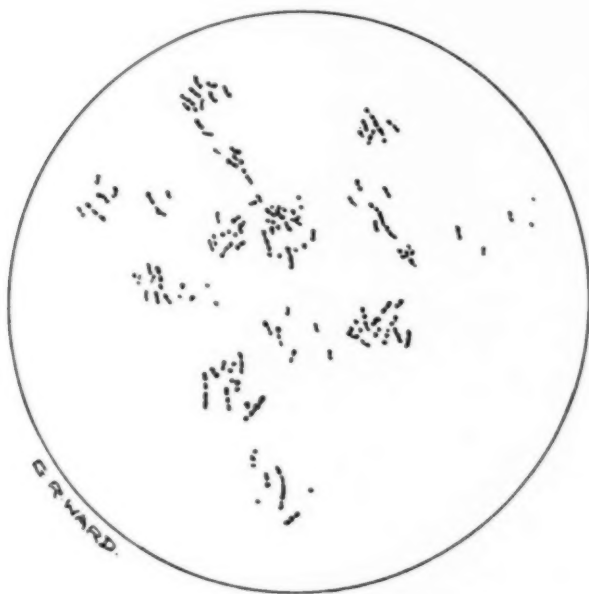


FIG. 5.

Ten days' culture in broth, showing disappearance of the filaments.

much swollen,¹ so it was killed next day. Nodules were found along the parietal peritoneum and a few scattered along the visceral membrane; a large caseating area on the under surface of the liver; fine nodules on

¹ It was at first thought that the lesions in the scrotum were due to *Bacillus mallei*, which was being used at the time for experimental purposes; but the possibility of the cultures having been confounded was put out of court by the subsequent experiments with repeated production of scrotal lesions. Hence this similarity of results is of great diagnostic import.

the gall-bladder and some caseating material united the layers of the tunicae-vaginales, whilst the testicles were enlarged. Lungs and heart normal. The pus was white and caseous, and contained filaments and numerous acid-fast spore-like bodies in groups. The filaments showed marked beading as described.

(3) Another guinea-pig was inoculated with a smaller dose of an agar-culture a fortnight after this, but as it appeared well six days later a second dose of a broth-culture was given into the peritoneum. The animal behaved like the previous ones, and on the third day was apparently moribund, with very large testicles; but it remained in this condition for a few days and then slowly recovered, the testicular swelling subsiding. It was killed some weeks later and no lesions of any note found. The animal at one time was so ill and had emaciated so rapidly that it seemed impossible for it to recover.

(4) Later two animals were inoculated, the one intraperitoneally, the other subcutaneously, with some of the original pus that had been stored in sealed tubes for about ten weeks and from which the streptothrix was obtained (at the time of inoculation) in pure culture, but the animals remained well.

Rabbits.—A rabbit received about 0·75 c.c. of the original pus into an auricular vein, several punctures being made, and it was given a second dose a week later. The temperature was raised a little, but there were no marked general symptoms. Around the sites of inoculation several caseating nodules formed and increased to the size of an almond nut and burst. From the caseating contents of these the streptothrix was obtained in pure culture. The swellings slowly resorbed and only some fibrous nodules ultimately remained. The temperature remained at about 103·6° F. for some weeks, and the animal lost a little in weight, which was the more striking as it was not full-grown. It was killed in about two months and there was found nothing but some scattered miliary nodules in the lungs. These, histologically, were mostly degenerate and structureless and surrounded by fibrous tissue, but some consisted of round-cells and epithelioid-cells, amongst which no giant-cells could be found nor any micro-organisms. Evidently, whatever lesions had resulted from the inoculations were rapidly being removed.

(2) Another rabbit recently received 0·75 c.c. of a thick emulsion of a very old subculture into the auricular vein. It rapidly became very ill and died in fifty-four hours. The lungs and pleura were intensely inflamed, and scattered throughout the lungs and kidneys were numerous pin-point white areas. The organism grew with great

rapidity in the tissues, and microsections of the lungs and kidneys showed a *prolific mycelium growing from the surface of the pleura*, in the lungs and in the kidneys, forming a most striking picture.

(3) Another rabbit received 0.75 c.c. of an emulsion of a recent subculture, intraperitoneally. The temperature varied between 102° F. and 104° F., but there was no loss of appetite or marked general symptoms. It was killed in two weeks, and there were found caseous nodules on the mesentery and omentum, some the size of a small nut, and a few miliary nodules on the surface of the liver and on the mesentery. In none of these nodules were there any giant-cells found, but merely round-cells, central degeneration, and peripheral encapsulation; filaments of streptothrix were found.



FIG. 6.

Heart of rabbit opened to show the white vegetation on the tricuspid valve. Two smaller ones on the chordæ tendineæ; the top of the larger vegetation is blood-stained and therefore indistinct. (The specimen is preserved in the Westminster Hospital Museum, No. 659.)

(4) Another rabbit received on the same date as (3) 0.75 c.c. of the same culture into an auricular vein. No ill-health followed, but the temperature varied between 105° F. and 103° F., though caseating, pea-size nodules appeared at the site of inoculation. It was killed in three weeks and presented interesting appearances as follows: Recent pleurisy; on the right side the diaphragm was adherent to the pleura by means of a thickish, organizing, gelatinous exudate. Discrete miliary, caseous

nodules scattered throughout the lungs. In the heart was found a *large white vegetation*, pendulous and about $\frac{3}{8}$ in. long, attached to the tricuspid valve, and, near by, two minute vegetations of the same nature on the chordæ tendineæ (fig. 6). Miliary nodules in the kidney. The right epididymis was swollen and caseous, and from the pus streptothrix was obtained.

Mice.—Repeated inoculations, intraperitoneally and subcutaneously, into mice produced no results.

SUMMARY.

A man died of an acute suppurative pneumonitis affecting one lung, together with a granulomatous peritonitis. From the abscesses a streptothrix (branching, beaded and pleomorphic) was isolated. This grew well on artificial media, producing a characteristic white surface growth on bouillon, was Gram-positive and to a certain extent acid-fast, the spore-like bodies, which were also a characteristic feature, being the more acid-fast. It was pathogenic to guinea-pig and rabbits, but more so in the former, and not at all to mice. It was found in the tissues and in cultures from the lesions of the lung of the man and the lesions in the animals. These lesions consisted of abscesses and caseating nodules, which resorbed, and in the guinea-pig minute miliary nodules on the peritoneum. In the guinea-pig enlargement of the testicles was a peculiar feature. It is difficult to say definitely what the miliary granulomata on the peritoneum of the patient were. As nothing but an acid-fast bacillus was found in them, and as this would do for a tubercle bacillus or a streptothrix, and more suggestively for the former, the question cannot be decided until similar lesions are produced experimentally by this streptothrix. In the nodules produced on the peritoneum of the guinea-pig no giant-cells were found, but otherwise they resembled the human lesions. Further, Stuart McDonald [4], working with an organism resembling in most points this here described, produced in rabbits, on intraperitoneal inoculation, in three weeks disseminated nodules in the lungs containing giant-cells. Flexner [2] also, with a different streptothrix, produced, experimentally, nodules with giant-cells corresponding to those in tuberculosis.

The mode of infection in this case was not evident. The man was a carpet porter and spent much of his time in cutting up cocoanut matting, but it seemed impossible to attempt to isolate a streptothrix from this source. Streptotrichosis is far from uncommon, and numerous

cases are on record of infections in all parts of the body—the thorax, abdomen, brain, external parts, and generalized infections.

The literature has reached enormous proportions, and in a recent publication [5] an exhaustive bibliography includes references to over 1,500 papers on the subject. In this country Foulerton [3] and Dean [1] have treated the subject at length, and the former has attempted a classification on a biological basis. Of the many varieties that are known, it seems probable that those pathogenic to man can be included in a few broad groups. The organism described in this paper is in most respects similar to that described by Eppinger, and ought to be classified with it.

The specimens are preserved in the museums of the Westminster Hospital Medical School and the Royal College of Surgeons.

For permission to make use of the clinical notes I am indebted to Dr. Murrell.

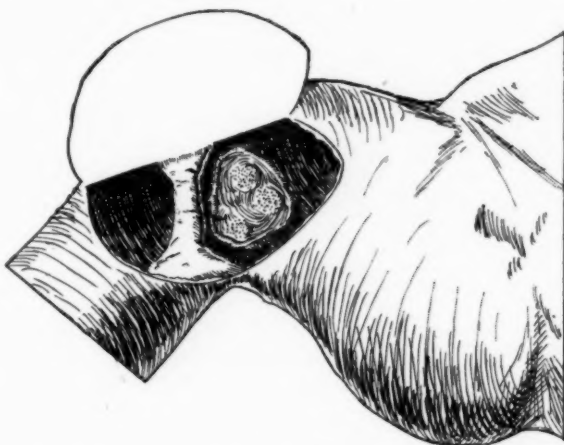
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- [1] DEAN. *Trans. Path. Soc. Lond.*, 1900, li, p. 26.
- [2] FLEXNER. *Journ. Experim. Med.*, New York, 1896, iii, p. 435.
- [3] FOULERTON. Allbutt and Rolleston, "System of Medicine," 2nd ed., 1906, ii, pt. i, pp. 302-24; *Trans. Path. Soc. Lond.*, 1902, liii, p. 56.
- [4] McDONALD. *Trans. Med.-Chir. Soc. Edin.*, 1903-4, N.S., xxiii, p. 131.
- [5] MUSGRAVE and CLEGG (with Bibliography by MARY POLK), *Philippine Journ. of Sci.*, Manila, 1907, ii, p. 477.

Congenital Malformation of the Pylorus.

By J. M. BERNSTEIN.

THE specimen here described consists of the stomach and pylorus removed post-mortem from a woman, aged 27, who, since the age of 17, had repeatedly attended Westminster Hospital with gastric symptoms which were ascribed to a peptic ulcer. In July, 1907, the symptoms became worse, being of the nature of fortnightly attacks of "pains across the back and stomach," having no relation to food but exacerbated by exertion; vomiting and localized pains, but no dilatation. A few days



Stomach and commencement of duodenum; a flap has been raised from the anterior wall of the pylorus to expose the band of tissue dividing the lumen into anterior and posterior channels; in the posterior wall is seen a chronic ulcer with the exposed pancreas as its floor.

later a laparotomy was performed and revealed a blind tubular diverticulum springing from the pyloric end of the stomach near the lesser curvature and ending blindly at its outer end, where it was attached to the anterior abdominal wall. This mass, the size of the terminal joint of the index finger, appeared to take origin from the site of an

old ulcer. Four months later she returned complaining of pains after food, occasional nausea and vomiting, and abdominal pain and back-ache, especially on exertion; sometimes food relieved the pain and sometimes vomiting. A gastrojejunostomy was performed, but the patient died soon after.

Post-mortem findings: There was some attempt at hour-glass formation owing to some cicatrization around the site of operation for removal of the ulcer, at which spot indeed there was found a smooth-walled ulcer, the pyloric portion being in the form of a sac. The pyloric ring was divided by a band of tissue, cylindrical in form, about $\frac{3}{8}$ in. in diameter, running a little obliquely so as to divide the pylorus into an anterior larger aperture and a smaller posterior one. The band was covered with normal mucosa and there was no evidence of inflammation in or around the band, which to all appearances was of congenital origin. On the posterior wall of the stomach at this spot—that is, in the floor of the lesser channel—was a chronic ulcer, $\frac{3}{8}$ in. in diameter, lying in both the duodenum and the pylorus, and having as its floor the exposed pancreas.

Histological examination shows that the band is exactly at the junction of the duodenum with the pylorus, the band itself being covered with pyloric mucosa (the gastric side of the band was alone examined). All the remaining viscera were normal save the left kidney, which was of small size, weighing only 2 oz., and had a depressed scar running round the entire meridian; the cause of this was not evident, the arteries being normal. The right kidney weighed 6 oz.

As far as one can judge, it would appear that this peculiar division of the pylorus was of congenital origin, and, further, caused no interference with the normal physiological processes, seeing that no gastric symptoms occurred until the age of 17, and these could well be accounted for by the chronic ulceration. I have been unable to find any case resembling this reported in the literature as far as I have gone into it.

For the illustration accompanying this description I am indebted to Mr. G. R. Ward, of the Westminster Hospital. (The specimen has been sent to the Museum of the Royal College of Surgeons.)

A large Laryngeal Lipoma of the Epiglottis and Base of the Tongue, with a Collection of Examples of Submucous Lipomata of the Intestine and of the Larynx.

By S. G. SHATTOCK.

THE very unusual position of this tumour, and its large size (for the situation in which it has grown), may render it worth recording from its anatomical side; that it was the cause of sudden death invests it, moreover, with a clinical interest. The specimen itself I have thus described in the pathological catalogue of the museum of the Royal College of Surgeons, to which museum it has recently been added:—

“The posterior part of a tongue with the larynx and portion of the pharynx. Growing to the left of the middle line, partly beneath the mucous membrane of the front of the epiglottis, and partly beneath that of the base of the tongue, there is a large, almost spherical lipoma. The tumour is smooth and polished on the surface, and measures $1\frac{1}{2}$ in. in its greatest diameter, but less at its base of attachment, so as to be rather pedunculated than strictly sessile. Its area of attachment to the tongue and epiglottis corresponds with the space between the median and the lateral glosso-epiglottic fold of the left side. Histologically it consists of perfectly normal adipose tissue, and is directly covered with the mucous membrane, which is itself furnished with a normal stratified epithelium.”

A certain number of the nuclei of the fat-cells show a single conspicuous vacuole of varying size. These vacuoles occur regularly in normal fat, and are due, as I have shown by staining sections of the latter with Sudan III,¹ to a fatty infiltration of the nucleus itself, like that of the body of the cell. When such a nucleus is viewed from the side, or edgewise, it is seen to project beyond the general contour of the vesicular cell like the boss of a shield. In optical section the intranuclear droplet is found to be almost spherical, with its greater convexity outwards; it does not bulge towards the cavity of the cell, the tension within which prevents any encroachment

¹ *Trans. Path. Soc. Lond.*, 1903, liv, p. 216.

on this aspect; at either side it is sharply bounded by the nuclear chromatin which rises up for a varying distance around it.

EXPLICATIO FIGURAE.

Monstratur lipoma magnitudinis insignis e linguae dorso epiglottideque ortum. Subito decessit aeger, tumore forsitan in gulam dislocato. Magnitudinis naturalis.



A view of the larynx from behind, showing the lipoma described; it projects from the front of the epiglottis and base of the tongue, on the left side of the median glosso-epiglottic fold. (Museum of the Royal College of Surgeons.) Natural size.

The parts were taken after death from a man aged 76. The history of the patient was, that after entering a restaurant he made an incoherent noise and a motion with his hands which was taken to be a

request for water. Death took place quite suddenly. At the autopsy the right lung showed marks of old inflammation; the heart was slightly enlarged; the aortic valve much diseased. No history of the patient, who was a pedlar, was obtainable, beyond that when calling at the police station a month before his death it was noticed there that he spoke in a very unusual way and was difficult to understand. The donor of the specimen, Mr. J. C. Rix, of Tunbridge Wells, who made the post-mortem examination, the case being the subject of an inquest, took the swelling, from its softness, to be a cyst.

The immediate cause of death was probably a displacement of the tumour. What apparently happened was that from some unusual act on the part of the patient the growth became suddenly engaged in the grasp of the pharyngeal constrictors and, together with the epiglottis, was tightly impacted over the upper aperture of the larynx; the patient, in short, involuntarily attempted to swallow his tumour.

Fatty tumours, in addition to such as are subcutaneous, or sub-fascial, intermuscular, periosteal, or parosteal, may be grouped as sub-mucous, subserous, and subsynovial. The present specimen falls, of course, into the submucous group. Submucous lipomata have been met with in very varied positions, and some, like those of the intestine, are of marked surgical importance. Two such I have added to the museum of St. Thomas's Hospital; both were removed by surgical operation. The fatty tumours growing from the peritoneal aspect of the bowel have no clinical interest, for they produce no symptoms of any kind; with the submucous lipomata which project as polypi into the lumen the case is different. Both Rokitansky and Virchow refer to submucous lipomata of the stomach and intestine.

Virchow¹ figures a small lipomatous polyp projecting into the stomach near the pylorus, and one of larger size, in another case, projecting into the jejunum; he alludes also to the colon as a further site of such tumours. In explanation, he observes that one will never examine the body of a well-nourished person without finding fat-cells in certain spots of the mucosa of the stomach or intestine.

In a case recorded by Sangalli (cited by Virchow) two submucous pedunculated lipomata were present in the descending colon; each was the size of a hen's egg, and their presence had induced invagination and prolapsus.

¹ "Die Krankhaften Geschwülste," Berl., 1863, i; Lipome, pp. 364-95.

Hillier has, in an article in Bruns's "Beiträge,"¹ collected the examples recorded in continental literature, but, with the exception of a reference to the work of Sir Frederick Treves² ("Intestinal Obstruction, 1899"), the cases described in our own language are omitted from Hillier's list. To Hillier's list of submucous lipomata of the intestine Langemak has added two further.³

I may supply, therefore, the original cases in our own literature, since they are not referred to by the continental authors named.

SUBMUCOUS LIPOMATA OF THE INTESTINE.

Coupland, 1879.⁴

This case was one of intussusception of the ileum into the cæcum through the ileo-cæcal valve, associated with a lipomatous polypus. Laparotomy was performed on the fifth day, the intestine being opened and secured externally above the seat of obstruction, since reduction of the intussusception was found to be impossible. Death occurred two days later. At the autopsy, the upper 3 in. of the central tube was found occupied by a firm, fleshy, cylindrical polypoid mass, almost the size of the little finger. The author remarks that the existence of a polypoid lipoma, and its situation at the part of the bowel the last to become invaginated, were points of interest; and that the presence of the tumour doubtless prevented reduction as well as hastening the ulceration which was present.

Stabb, 1894.⁵

Of the two examples in the museum of St. Thomas's Hospital, the first (1107 F) is a coarsely lobulated submucous lipoma which was removed from the ileum. In the pedicle there is a small portion of the proper muscular coat of the bowel, slightly prolapsed by the traction of the tumour. Over the most prominent parts of the growth the mucous membrane has been destroyed by ulceration.

¹ "Über Darmlipome," Bruns, *Beitr. z. klin. Chir.*, Tüb., 1899, xxiv, p. 509.

² The specimen referred to by Sir Frederick Treves is no longer in the museum of the London Hospital, but he writes to me that it showed the bowel in section and a single polypoid ingrowth.

³ Bruns, *Beitr. z. klin. Chir.*, 1900, xxviii, p. 247.

⁴ *Brit. Med. Journ.*, 1879, i, p. 854.

⁵ *St. Thomas's Hosp. Reports*, (1894) 1896, xxiii, p. 115.

The patient, aged 32, was admitted under the care of Dr. Ord, March 5, 1894, and was operated upon by Mr. E. C. Stabb. On the morning of admission he had been suddenly seized with acute pain in the lower part of the abdomen, accompanied with violent vomiting. A rounded movable swelling could be distinguished in the lower abdominal region extending into the pelvis. After median laparotomy, the mass previously felt was found to be an intussusception of the small bowel; reduction of this was effected by slight traction upon the entering tube, after which a small, irregular, pedunculated tumour could be felt within its lumen about 30 in. from the cæcum. The growth was removed with an elliptical piece of the intestinal wall to which the pedicle was attached. The incision in the gut was closed with a double row of Lembert's sutures of fine silk. The patient progressed well until the ninth day, when the temperature suddenly rose. A small area of dullness could be detected just to the left of the middle line and below the umbilicus; on incision a small cavity containing fæcal matter was opened, and a small slough was seen in the bottom of the space, protruding from an aperture in the intestinal wall. On gently pulling this a long slough came away, followed by fæcal discharge; the wound was consequently in part left open, and a fæcal fistula resulted. Ten days later extensive hæmorrhage occurred within the bowel, the blood appearing through the abdominal fistula; a second hæmorrhage ensued, and the abdomen was opened, a matted bunch of small intestine about the size of a cocoanut, and into which the fistula opened, being resected. This mass probably included the whole length of bowel originally involved in the intussusception. The patient became subsequently collapsed, and died. On slitting up the bowel removed it was found much reduced in calibre, and no healthy mucous membrane anywhere remained, the surface being almost throughout ulcerated. The lumen was full of recent clot, but no vessels could be found from which the hæmorrhage had taken place.

Shattock, 1899.¹

The second specimen (No. 1107 H, St. Thomas's Hospital Museum) is an oval lipoma $1\frac{1}{2}$ in. in chief diameter, and was removed from the interior of the sigmoid flexure. The chief part of the growth is covered with mucous membrane, in which there is a circular defect indicating the position of the divided pedicle. Over the most prominent part of the tumour the mucosa has been destroyed by ulceration.

¹ Pathological Catalogue, St. Thomas's Hospital Museum; S. G. Shattock.

The patient was a man, aged 36, who was admitted under the care of Dr. Payne in October, 1899. He had been seized with abdominal pain on October 20. Constipation was absolute; blood was noticed by the rectum; there was tenesmus, but no vomiting or distension of the abdomen. An enema brought away only blood. On October 31 an incision was made by Mr. William Anderson in the left iliac region, the sigmoid flexure opened, and the lipoma removed after ligature of its pedicle. The wounds in the gut and abdominal wall were closed. Complete recovery ensued.

Bland-Sutton, 1900.¹

Mr. J. Bland-Sutton has also successfully removed from a man, aged 44, a lipoma weighing 2 oz., which occupied the submucous tissue of the ascending colon above the ileo-cæcal valve. The patient had passed through several acute attacks of intestinal obstruction.

Knaggs, 1900.²

In this case enteric intussusception resulted from the presence of an intestinal lipoma. The patient was a woman, aged 29. The intussusception was reduced, and the lipoma, which was covered with the intestinal mucosa, was removed by an incision carried through the wall of the gut.

In the museum of St. Bartholomew's Hospital there are two specimens of submucous lipoma of the small intestine which were found (in different cases) accidentally after death. Neither measures more than 2 cm. in chief diameter, and each projects as a polypus. One is attached to the second part of the duodenum; the other to some part of the "small intestine."

And, to close the list; quite recently at the autopsy of a man, aged 40, in St. Thomas's Hospital, a Meckel's diverticulum, 6 cm. in length, was removed, to the blind end of which there was attached an oval polypus 2·3 cm. in length and 1·2 cm. in breadth. The tumour was smoothly covered with mucous membrane, and closely filled the terminal part of the diverticulum. On dividing the growth I found it to be a pure submucous lipoma. The patient had suffered at intervals throughout his life from attacks of abdominal pain, associated with vomiting and

¹ *Lancet*, 1900, i, p. 1437.

² *Ibid.*, 1900, ii, p. 1573.

constipation, which usually cleared up at the end of a week. When admitted, he had been suffering from such an attack for fourteen days. On abdominal exploration a Meckel's diverticulum was found passing from the small intestine in the right iliac region upwards to the lower surface of the liver, to which it was adherent. The terminal 3 in. was cord-like. For its lower 3 ft. the ileum was much coiled upon itself, bound by adhesions, and in some manner strangulated by the diverticulum. An ileostomy was carried out by Mr. Battle. Death took place a week later from exhaustion. The patient was rather sparely built, and was certainly not obese.

As an example of submucous lipomata of the mouth I may adduce one of a spherical tumour, about $1\frac{1}{2}$ in. in diameter, which was removed by Mr. Walter Edmunds from beneath the mucous membrane of the lower lip (No. 256 B, Museum of St. Thomas's Hospital). And, as showing how history repeats itself, Lebert has referred to a similar tumour of the size of a small nut in the same position.¹ On more than one occasion ranula has been closely simulated by a lipoma beneath the tongue. Marjolin (Virchow, loc. cit.) saw a submucous lipoma in the floor of the mouth which suggested this condition. In the museum of the Middlesex Hospital, also, there is a fatty tumour which was removed from beneath the tongue, where it looked like a ranula (Paget, "Lectures on Surgical Pathology: Fatty Tumours"). And in the museum of the Royal College of Surgeons there is a discoidal lipoma, 4 cm. in chief diameter, which was removed from under the tongue (Specimen No. 318). There is a wax cast of the same specimen in the museum of St. Thomas's Hospital. But to come more particularly to the lipomata which, in different degrees, like that recorded in this communication, involve the larynx. In our own literature there are at least two worth citing.

LARYNGEAL LIPOMATA.

(1) S. Jones.²

One of these is a deeply lobulated, somewhat flattened lipoma, about $2\frac{1}{2}$ in. in diameter, which was removed from the right aryepiglottic fold of a man, aged 40. The tumour hung down into the pharynx, and could be protruded at will into the patient's mouth (Specimen No. 1786 B,

¹ "Traité d'Anatomie pathologique," 1857, p. 125.

² S. Jones, *Trans. Path. Soc. Lond.*, 1881, xxxii, p. 243.

St. Thomas's Hospital Museum). An incision was made through the mucous membrane and the mass was at once turned out.

(2) Holt.

The second case is that reported in the *Transactions of the Pathological Society* by Mr. Holt (vol. v). It concerned a man aged 80, and is recorded as a "fatty pendulous tumour of the pharynx and larynx." There was a twelve years' history of a sensation of choking, which became more frequent, and the patient was aware of some swelling, or slight bulging, at the upper part of the throat. About four years before his death a large mass became protruded during the act of vomiting, and to prevent immediate suffocation he was compelled to return it as quickly as possible. He was better able at all times to swallow solids than fluids. In swallowing fluids he occasionally experienced great difficulty and choking, but latterly, by taking everything very slowly, he was comparatively comfortable. His voice was husky though occasionally distinct, more especially if he was perfectly calm; but, when excited, it became gurgling and inarticulate. He died suddenly while smoking his pipe, and it was conjectured (there being no one present) that the tobacco smoke, by producing sudden cough, led to displacement of the growth and to immediate suffocation. At the autopsy a large, pendulous, fatty tumour was found filling the pharynx and extending downwards in the œsophagus for a distance of 9 in. It was smoothly covered with mucous membrane, and was attached to the left aryepiglottic fold (the epiglottis being displaced downwards on the left side) and to the anterior wall of the pharynx, chiefly over the posterior aspect of the cricoid cartilage; beyond this the tumour was free, of cylindrical form, and filled the œsophagus below for several inches. The naked-eye diagnosis was confirmed by microscopic examination. An excellent plate accompanies the account.¹

In the *Proceedings of the Laryngological Society*, January, 1902, Dr. Milligan records a specimen of pharyngeal lipoma which was successfully removed from a woman, aged 37, who had complained of slight dysphagia and a feeling of fullness in the throat, and a considerable amount of dyspnoea when lying down. Upon examination a large unilateral ovoid swelling was found under the mucous membrane of the posterior wall of the pharynx on the left side. It extended upwards

¹ Meckel reports a case in which a submucous lipoma occupied the lower extremity of the œsophagus (Virchow, loc. cit.).

behind the level of the soft palate and downwards behind the larynx, where the swelling was most prominent. The growth was removed from outside through a lateral incision. Although the tumour in this case produced laryngeal symptoms, it cannot be classed as a laryngeal lipoma.

Schrötter,¹ in his text-book, refers to two cases, observed by Wagner and Tobold, pointing out that in the second of these the lipoma hung into the larynx without being strictly laryngeal in its attachment.

(3) Wagner.²

Wagner's example was one observed at the autopsy of a patient dying of smallpox. The lipoma was the size of a hen's egg. The patient, a girl, aged 13, was to have been operated upon. The growth was attached by a thin broad fold in the mid-line to the dorsum of the tongue and front of the epiglottis; it was flattened from behind, elsewhere lobulated, and was freely movable.

(4) Tobold.

Tobold's case³ was that of a man, aged 64. The upper laryngeal aperture was occupied by a tumour as large as a walnut. Death occurred from septic bronchitis five days after the performance of tracheotomy. Examination showed the origin of the lipoma to be from the wall of the pharynx about the level of the arytenoid cartilage. It was smooth and pale in colour, and was pedunculated so as to lie like a ball-valve over the aperture of the larynx.

(5) Bruns.

In the case recorded by Bruns the tumour arose from the hinder wall of the larynx over both arytenoid cartilages, and most probably was congenital. Microscopic examination after its removal demonstrated its fatty nature.

(6) Schrötter.

Schrötter contributes a case of his own, with two accompanying figures (*loc. cit.*) The tumour here involved the middle glosso-epiglottic

¹ "Krankheiten der Kehlkopfes," Wien, 1892, i, p. 270.

² E. Wagner: "Die Todesfälle in der letzten Pockenepidemie von Leipzig," *Archiv der Heilk.*, Leipz., 1872, p. 108.

³ "Laryngoscopie," Berl., 1874, 3 Aufl., p. 436.

ligament, the lateral border of the epiglottis at the attachment of the pharyngo-epiglottic ligament, and the whole length of the left ary-epiglottic fold. Digitate processes extended partly into the larynx, partly into the sinus pyriformis, and partly into the space between the hinder laryngeal wall and the pharynx. Microscopical examination of the tumour after its removal showed it to be a lipoma arising in the submucous tissue.

Seifert and Kahn ("Atlas," Wiesbaden, 1895) refer only to two examples of what they speak of as "Die äusserst seltenen Lipoma des Kehlkopfes."

(7) Hohlbeck.

One of these is recorded by Hohlbeck,¹ whose figure is therein reproduced. It shows a deeply lobulated lipoma surrounding the upper aperture of the larynx, the tumour growing from both the aryepiglottic folds, but anteriorly projecting up in front of the epiglottis from the base of the tongue, the epiglottis itself being fairly intact. The growth as a whole is annular, but the ring is incomplete in the situation of the interarytenoid fold. The subdivision of the tumour is hardly sufficiently complex to justify the designation "arborescens" adopted by its describer.

(8) Seifert and Kahn.

In the second example the tumour sprang from the hinder wall of the larynx; its histology is given by the authors of the "Atlas." The limit of the adipose tissue in this case is not very sharply defined. Towards the middle of the tumour the fat is arranged in lobules of different sizes; close beneath the epithelium (of the stratified squamous kind) the connective tissue, which preponderates at the periphery of the tumour, is strewn with small groups of, or with single, fat-cells.

The actual site of the growth in the case recorded in the present communication presents no difficulty in regard to its histological explanation, since as the common form of fat is merely connective tissue of which the cells are infiltrated with oil, a lipoma may grow in any position, for connective tissue is practically ubiquitous. Selecting the body of an obese woman, aged 36, who had died of chronic nephritis, I examined the following laryngeal sites for the presence of fat-cells as a normal phenomenon:—

¹ "Ueber Lipoma arborescens des Kehlkopfes."

(1) Coronal or transverse vertical section of the base of the tongue, close in front of the epiglottis: Groups of fat-cells occur between the mucous glands; and horizontal groups lie above these in the connective tissue of the deeper part of the corium, the corium itself being undemarcated from the subjacent substance of the tongue.

(2) Horizontal sections of the interarytenoid fold, studied from the posterior or pharyngeal aspect: Groups of fat-cells occur between the numerous mucous glands which lie under the corium, and other groups occur superficially to the glands in the deeper part of the corium. On the deep side of the glands collections of fat-cells are present on the subjacent muscular tissue, and between its more superficial fasciculi.

(3) Horizontal sections of the pharyngeal mucosa and subjacent posterior crico-arytenoid muscles at a level of the middle of the back of the cricoid cartilage: Horizontal groups of fat-cells occur in conspicuous numbers in the deeper part of the mucosal connective tissue close above the surface of the subjacent muscles, and, in fewer numbers, fat-cells occur between the two muscles in the connective tissue over the median portion of the cricoid cartilage. Sections similar to the three foregoing made from an obese male, aged 42, who died of cerebral hæmorrhage, show a precisely similar distribution of fat.

(4) Aryepiglottic fold. Vertical antero-posterior sections carried in the long axis of the fold: In front of the arytenoid cartilage there is a close cluster of mucous glands, in the connective tissue between which, and between these and the surface-epithelium, there are a few stray fat-cells. The connective tissue in front of this collection of glands contains small groups of fat-cells; one small group lies at no great depth from the epithelium, and a short way in front of this there is a second group; more deeply in the connective tissue the collections are larger, and in the deepest part, where the muscular fibres of the aryepiglottideus are reached, the groups of fat-cells are considerable. A corresponding section made from the larynx of the obese male before referred to shows a similar disposition of fat, considerable groups of fat-cells occurring between the fasciculi of the muscle, and smaller groups between the muscle and the free edge of the fold at no great distance from the investing epithelium.

(5) Sagittal section carried through the base of the tongue and the epiglottis between the median and lateral glosso-epiglottic fold: On the posterior surface of the epiglottis, small collections of fat-cells occur in the immediate neighbourhood of the elastic cartilage, but not in the neighbourhood of the apex; a few of the fat-cells occur in connexion

with the mucous glands which lie in close relation with the cartilage; and some such glands of flattened, compressed form are present not far from the apex. On the anterior, lingual aspect, the fat is in greater amount. Extensive flattened groups of cells occur in the immediate neighbourhood of the cartilage in interrupted series, for the whole length almost as far as the apex; towards the base, where the mucous glands are in evidence, groups of fat-cells occur in relation with the glands—*i.e.*, around them and in the interglandular connective tissue. In the bottom of the fossa between the tongue and epiglottis there is quite a notable amount of fat in the connective tissue beneath the mucosa; this fat is continued forwards into the muscular substance of the tongue.

In the obese male before mentioned sagittal sections of the epiglottis made close to one side of the mid-line show, on the posterior aspect of the cartilage, groups of fat-cells for the whole length, reaching as far as the apex of the cartilage, as well as in the substance of the mucous glands where these occur; the glands are absent in the immediate region of the apex. On the anterior aspect, groups of fat-cells occur in the mucous glands; and towards the apex, unconnected with glands.

In order to ascertain if fat was present in the base of the tongue of the patient from whom the epiglottic tumour was taken, I cut microscopic sections in a coronal or tranverse vertical plane within a short distance of the site of the lipoma. The sections display horizontal rows or flattened groups of fat-cells in the deeper part of the corium, and superficial to the glands in this position. There are no fat-cells in the papillæ; other fat-cells or cell-groups occur in the connective tissue of the glands themselves, and others between the fasciculi of the muscular substance.

It will appear, therefore, that fat-cells may be normally met with in all the positions from which these different laryngeal lipomata have been found growing. And, in conclusion, it may be pointed out that all the tumours hitherto recorded fall into the extrinsic group.

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The Council think it right to state that the Society does not hold itself in any way responsible for the statements made or the views put forward in the various papers.

Surgical Section.

October 13, 1908.

Mr. J. WARRINGTON HAWARD, President of the Section, in the Chair.

Traumatic Rupture of the Intestine, with a Case of Recovery after Operation and an Analysis of the 132 cases that have occurred in ten London hospitals during the last fifteen years (1893-1907).

By JAMES BERRY, F.R.C.S., and PAUL L. GIUSEPPI, F.R.C.S.

WILLIAM H., aged 26, was admitted to the Royal Free Hospital under the care of Mr. Berry on September 25, 1907, suffering from the effects of a kick on the abdomen. On September 25, 1907, at 5 p.m., the patient received a kick just above the pubis from a horse close to which he was standing. He was not knocked down. He immediately felt severe pain in the hypogastrium, and was brought to the hospital in a cab at 5.15 p.m. On examination, a slight bruise was found just above the pubis. There was tenderness over this area. The abdomen moved with respiration. The patient was not collapsed and did not seem very ill; the pain slowly increased in severity, as did also the hypogastric tenderness. The abdominal muscles were markedly contracted and hard. He vomited several times. The bladder had been emptied four hours previous to the accident. Urine was passed twice after admission; on neither occasion did it contain any blood. He had had his last meal about 12 o'clock. A severe injury to the abdomen and probably ruptured intestine was diagnosed, and Mr. Berry summoned.

Shortly before 9 p.m. the patient was first seen by Mr. Berry. There were then severe abdominal pain and marked tenderness over the hypogastrium. The muscles of the abdominal wall were very rigid,

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and there was some dulness in the left lumbar region in front. The liver dulness was normal. *Per rectum*, nothing abnormal could be felt. The pulse-rate had slowly risen from 76 to 96, temperature was 99° F., respirations 26. It was decided to perform a laparotomy as soon as possible. The operation commenced at 9.45 p.m. and lasted twenty-five minutes, chloroform and ether being administered; m^{v} of liquor strychninæ were injected before the operation began. An incision 6 in. long was made $\frac{1}{2}$ in. to the left of the middle line, commencing 1 in. above the umbilicus. On opening the peritoneal cavity no gas, but a thin, non-odorous, gruel-like material escaped; in it portions of French beans were obvious. The visceral peritoneum had not lost its lustre, but was injected and slightly granular, and there were also some flakes of lymph adherent to the small intestines. A complete longitudinal rupture $1\frac{1}{2}$ in. long was quickly found on the side opposite to the mesenteric attachment. The mucous membrane was slightly everted. The affected coil was brought out, an intestinal clamp applied, and the contents squeezed out. The opening was closed with a double row of continuous fine silk sutures. The first row passed through all the coats; the second row was of Lembert sutures. The opening was closed in the direction of the long axis of the bowel. The lumen of the gut was reduced thus to about one-half its normal size. The rupture was situated in the ileum, judging from the size of the gut; no other rupture could be found. Saline solution at 105° F. was poured into the peritoneal cavity and then expelled by compressing the abdominal wall. This was repeated several times; the time thus spent did not exceed four minutes. A rubber tube, 8 in. long and $1\frac{1}{2}$ in. in diameter, was inserted into Douglas's pouch. The abdominal wall was united in two layers with catgut and silkworm-gut sutures. The patient was then turned on to his face for half a minute, with the shoulders raised, so as to empty the abdomen completely.

The patient, on returning to bed, was sat up at an angle of 45°. The after-treatment consisted in the subcutaneous administration of m^{v} of liquor strychninæ every hour and rectal infusions of saline (2 pints) every two hours.

September 26: Temperature 100° F., respirations 26, pulse 102; complexion sallow. The patient had had but little pain. He was now sat up erect in bed. At 10 a.m. the tube was removed and replaced by one $\frac{3}{4}$ in. in diameter. There was some difficulty in removing the tube, as a coil of small intestine had slipped into one of the holes in the tube. Some discharge was sucked out of the pelvis. The patient had

continued to vomit. Five minims of liquor strychninæ were now given every two hours, and saline 1 pint *per rectum* every two hours. September 27: Patient less sallow. The wound was dressed in the morning, and a large piece of membranous lymph was removed. There was hardly any discharge from the tube. There was some little distension of the abdomen, and the patient continued to vomit; the vomit had an intestinal odour. In the afternoon the tube was removed and the wound lightly packed with gauze. Leucocyte count, 15,200 per cubic millimetre; liquor strychninæ, mv , every two hours. Five grains of calomel were given with a good result, and a turpentine enema. September 28: The patient was much better and able to read and enjoy the morning paper. The vomiting ceased at 4 a.m.; the distension was less. Five minims of liquor strychninæ every six hours; 4 oz. of peptonized milk every two hours. September 29: Milk 6 oz. by the mouth every four hours was given to the patient, who up to this time had had nothing but water by the mouth.

Since admission the patient had absorbed 26 pints of water *per rectum* and had had 173 minims of liquor strychninæ subcutaneously; of these, 95 were given in the first twenty-four hours; he never had muscular twitching at any time. On the morning after the operation the temperature rose to 100.2°F .; after this it never reached 100°F ., and after the third day it remained perfectly normal. The highest pulse-rate recorded was 102 on the morning after the operation; after this it was generally between 80 and 90.

October 1: The wound looked healthy, the stitches were removed except those just above the sinus. For the last three days the bowels had been opened without any purgative. October 2: The remaining stitches were taken out. October 3: The edges of the wound gaped for $1\frac{1}{2}$ in. October 8: The sinus extended backwards for 2 in. October 15: Sinus measured $1\frac{1}{2}$ in. October 22: The sinus has completely healed.

November 4: The granulating wound was stitched up. November 12: The wound measured $1\frac{3}{4}$ in. wide. The stitches that were put in did not hold. November 14: The patient left for a convalescent home fifty days after the operation.

December 11: The wound had now completely healed. The scar seemed strong. A belt was ordered.¹

Leucocyte counts from September 27 till October 17 varied between 10,000 and 17,000.

¹ The patient was shown at the meeting on October 13. He was in excellent health and said that his bowels acted regularly and normally.

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This case has led us to an investigation into the subject of rupture of the intestine, in the hope that we might be able to throw more light upon the difficult problems of the early diagnosis of such injuries and the best methods of dealing with them. For this purpose we have collected notes of all the cases of ruptured intestine, without external wound, that have occurred in the last fifteen years (1893 to 1907 inclusive) in ten of the principal general hospitals in London (*see* Appendix, Table I). These hospitals are Charing Cross, Guy's, London, Royal Free, St. Bartholomew's, St. George's, St. Thomas's, University College, King's College, and Middlesex. To the various Surgical Registrars and other authorities of these hospitals we desire to express our grateful acknowledgment for the help afforded to us in obtaining access to these records. Many of the cases have been published already, in hospital reports or elsewhere, but most of them are to be found only in manuscript in the hospital records.

The list of 132 cases embraces, so far as we are aware, all the cases of ruptured intestine that have occurred at these hospitals within the period mentioned. Among the many excellent papers that have been written on the subject of ruptured intestine we should like to refer especially to that of Hertle, who has given a detailed list of 145 cases, compiled partly from the records of the Graz (Austria) clinic, partly from other German and Austrian clinics, and partly from the literature (mainly recent).

Curtis's list of 116 cases was published in 1887, and is compiled wholly from published cases, mainly American and English. Few, if any, of Hertle's cases are to be found in Curtis's list, and none of ours occurs in either of the two other lists. These three long lists of cases do not overlap to any appreciable extent, and may be used for purposes of comparison. Our own list of cases is, so far as we know, the only large collection of cases drawn exclusively from hospital records, and has therefore in some respects more value than lists compiled solely or mainly from published surgical literature, in which successful or remarkable cases are apt to attain undue prominence. Of Mr. Makins's well-known list of 21 cases occurring at St. Thomas's Hospital during the years 1889 to 1898, no fewer than 14 are necessarily also included in our own list.

It so happens that, of the whole number of 132 cases in our list, 10 have been under the care of one of us at the Royal Free Hospital. Of the 27 cases that occurred at St. Bartholomew's Hospital, 7 of the earlier ones came under the observation of one of us when

Surgical Registrar at that hospital in the years 1893 to 1897 inclusive, and the post-mortem examinations on most of these were made by him. Upon the clinical and pathological knowledge thus obtained, and upon the examination of the hospital and other records above mentioned, the conclusions in our paper are based.

ETIOLOGY AND PATHOLOGY.

Tables III, IV, and V show the relative frequency of the injury in the two sexes and at various ages. As might be expected, it occurs most commonly in boys and young men, whose occupations render them more liable to accidents from violence. Rupture of the intestine in women and children is caused most often by being run over or by falls. Of the 14 children under 10 years of age, in 10 the injury was due to being run over, in 2 to falls, in 1 to a kick, and in 1 to a blow. Of the 10 women 7 were run over, in 1 case a wall fell on the patient, and in 2 the injury was due to a fall.

TABLE III—SEX.

		Berry and Giuseppe (ten London hospitals, 1898 to 1907 inclusive)		Petry (Prague clinic and literature)		Hertle (Graz clinic and literature)		Makins (St. Thomas's Hospital, 1889 to 1898 inclusive)
Male	...	122	...	217	...	124	...	21
Female	...	10	...	15	...	4	...	0

TABLE IV—AGE (STATED IN 125 CASES).

Berry and Giuseppe.

Total cases	Under 5	5 to 10	10 to 20	20 to 30	30 to 40	40 to 50	50 to 60	60 to 70	Over 70	Not stated	Situation of rupture
10	—	—	—	3	3	2	—	1	—	1	Large intestine
23	—	5	7	4	1	1	3	1	—	1	Duodenum ...
3	—	—	3	—	—	—	—	—	—	—	Duodeno-jejunal flexure ...
32	—	1	8	10	4	4	3	—	—	2	Jejunum ...
32	1	2	6	4	3	5	6	2	—	3	Ileum ...
25	2	3	5	4	7	—	2	2	—	—	"Small intestine" ...
4	—	—	1	2	—	1	—	—	—	—	Large and small intestine
3	—	—	2	1	—	—	—	—	—	—	Partial rupture
			(1)	(1)							
132	3	11	32	28	18	13	14	6	—	7	

Figures in brackets mean recoveries.

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TABLE V—HERTLE'S COLLECTED CASES.

Age	Men	Women
0 to 10 years	5	—
11 " 20 "	14	—
21 " 30 "	33	—
31 " 40 "	35	—
41 " 50 "	27	1
51 " 60 "	17	2
61 " 70 "	3	1
Not stated	7	—
	141	4

Hernia to a small extent predisposes to rupture of the intestine, as shown in Case 87, in which a mass of lead fell upon an inguinal hernia and caused rupture of the intestine.

When we come to consider the causation of the injury, it will be seen at once that the various forms of injury may be classed under very few headings, namely: run over, squeeze or crush, blow, kick, and fall. These five headings include almost all the cases in our series and in that of Hertle.

TABLE VI—CAUSES OF TRAUMATIC RUPTURE OF THE INTESTINE.

	Berry and Giuseppe (hospital records)	Hertle (literature)
Run over	51	10
Squeeze or crush	24	11
Blow	23	47
Kick	16	40
Fall	11	20
Reduction of hernia	1	—
Trodden on	—	4
Uncertain	6	—
Severe exertion	—	6
	132	138

As might be expected, our statistics, drawn from the hospital records of a crowded metropolis, show a relatively large number of run-over cases—no fewer than 51 out of 132. Hertle's figures, drawn mainly from literature, are 10 run-over cases out of 138; and Curtis's, taken also from literature, are 13 out of 116 cases. On the other hand, in only 16 of our cases was the injury due to a kick, while Hertle's and Curtis's figures are respectively 40 and 28. The discrepancy in these figures is probably due to the fact that ruptures due to kicks are less likely to be complicated by other severe injuries, are therefore more likely to be the subjects of operative interference, and consequently find their way more readily into surgical literature.

Of the 24 cases in our list in which the rupture of the intestine was complicated by other injuries, in no case was the injury stated to have

been due to a kick. Of the 24, no fewer than 19 were due to being run over or crushed, or to falls from a height.

The relative frequency of rupture of the intestine in cases of kick from a horse has been variously stated by different authors. Surgeon-Major Cahier, in an excellent recent article on injuries of the abdomen due to kicks, has shown that of 15 or 16 cases in which the injury seemed at first to be grave, in only 2 was laparotomy required for a lesion of the intestine. Pech collected 71 cases of injury to the abdomen from kicks, treated at the military hospital at Lunéville in twenty years, and found that 69 recovered without operative interference and only 2 died from ruptured intestine. On the other hand, civil hospital surgeons who deal with cases of kicks from horses probably see a larger proportion of severe cases than do the military surgeons.

The conditions under which the intestine is ruptured are almost invariably the sudden and violent impact of a hard substance against the bony portions of the posterior abdominal wall. The prominent vertical ridge of the lumbar spine and, to a less extent, the iliac bones are those against which the intestine is violently crushed. As regards the large intestine, a glance at Table VIII will show that it is precisely those portions that lie normally in front of these bones, namely the transverse colon, cæcum, and sigmoid flexure, that are most liable to injury. As Mr. Makins has pointed out, those parts of the intestine that are most exposed to injury are those which lie in the lower half of the abdomen. An important exception, however, is to be found in the duodenum, which, from its fixed position in front of the lumbar spine, is especially liable to injury. Of our 132 cases the duodenum was the part affected in no fewer than 24 (see Table VIII). It is remarkable that among Mr. Makins's 21 cases there should have been only one of ruptured duodenum.

TABLE VII—SITE OF RUPTURE.

	Berry and Giuseppe (ten London hospitals, 1898 to 1907)	Hertle (literature, recent)	Curtis (literature, older)
Duodenum ...	24	15	6
Duodeno-jejunal flexure ...	3	—	—
Within 3 ft. of duodenum or ileo-cæcal valve ...	27	—	25
Within 50 cm. (20 in.) of duo- denum or ileo-cæcal valve	—	28	—
Intervening small intestine ...	37	43	57
"Small intestine" (exact site unknown) ...	25	32	21
	116	118	109

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As regards the rest of the small intestine, our figures and those of Hertle and Curtis seem to show that there is some truth in the old belief that the parts of the small intestine that are most liable to injury are the upper part of the jejunum and the lower part of the ileum, that is, those parts that are comparatively near fixed points, have a shorter mesentery, and are more likely to be found lying in front of the lumbar spine and less likely to be able to slip aside and thus escape an impending blow. The rather large number of cases, however, in which the exact seat of rupture in the small intestine is not stated renders conclusions on this point of comparatively little value. All parts of the small intestine are undoubtedly liable to injury. The situation of the lesion in our series of cases is shown as far as possible in Table VIII.

TABLE VIII—SITUATION OF THE RUPTURE.

	Berry and Giuseppe		Hertle	
Large intestine :				
Cæcum	2		2	
Ascending colon	—		2	
Hepatic flexure	—		—	
Transverse colon	3		2	
Splenic flexure	1 ¹		—	
Descending colon	—		1	
Sigmoid flexure	4		3	
Rectum	—		1	
	10		11	
Small intestine only :				
Duodenum	23 ²		12 ³	
Duodeno-jejunal flexure	3		—	
Jejunum	32		29	
Ileum	32		40	
" Small intestine "	25		31	
	115 ⁴		112	
Large and small intestine	4		—	
Partial rupture (not involving lumen) :				
Large intestine	2		—	
Duodenum	1		—	
	3		—	
	132		123	

Of Hertle's series of 134 cases 20 were multiple.

Three theories as to the production of a rupture of the intestine have been brought forward, namely, that it is due to crushing, to bursting, and to traction. That direct traction is occasionally the cause of rupture is perhaps shown by the rather rare cases in which the tear

¹ This case was retroperitoneal.

² Three of these cases were retroperitoneal.

³ Seven of these cases were retroperitoneal.

⁴ Eighteen of these cases were multiple.

occurs at the duodeno-jejunal flexure, as in three of our cases (Nos. 64, 72 and 75). Bursting, we venture to think, is a rare cause of rupture; it can scarcely occur except when the intestine contains an unusually large amount of fluid. Hertle's series of interesting experiments on the production of rupture by bursting were carried out on living animals and dead human subjects. They show how the intestine can thus be ruptured. They are vitiated, however, in our opinion in that a preliminary ligature was applied to both ends of a coil of intestine prior to the application of violence. This condition of complete closure of both ends of a coil of intestine is one which, in our opinion, can seldom occur in the living human subject at a moment of injury. Hertle's experiments are of great value, however, in showing the kind of rent produced by bursting and crushing injuries respectively. In crushing injuries the muscular and mucous layers are those which are most easily damaged; the submucous layer is more resisting, and the peritoneum the most resisting of all. This is a point of great importance in connection with partial and secondary ruptures. All the coats of the intestine may be ruptured except the peritoneal or submucous, and the patient will then have few or no symptoms of injury. It is not until the peritoneal or submucous coat has given way secondarily that extravasation occurs and acute symptoms set in.

In Hertle's bursting experiments, on the other hand, it was shown that the submucous layer was usually the last to give way, the peritoneal being torn early. This is in accordance with what we so often see in extreme distension of the cæcum due to stricture, rents of the peritoneal coat often preceding the actual perforation into the lumen. A careful examination of the rent in the intestine will sometimes enable us to say whether or not the injury has been produced by bursting. For our own part we believe that rupture of the intestine in the human subject is almost invariably produced by direct crush, and not by bursting. One of the rare exceptions, Case 68, in which the rupture was caused by the violent reduction of a hernia, is probably one in which the rupture was produced by bursting. The examination of museum and post-mortem specimens, as well as our necessarily very limited experience on the living subject, appears to us to bear out this contention. It is to be regretted that the notes of hospital records are usually not sufficiently full to enable us to draw conclusions from them on this point.

Multiple ruptures are by no means uncommon, as will be seen from Table IX. In 22 of our cases the intestine was ruptured in two or more places. The ruptures are usually not far apart. In 44 of our

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cases the notes make mention of the direction of the wound in the intestine. In 38 of these it was transverse, in the remaining 6 it was longitudinal. Of the 38, in 15 the rent involved the whole circumference of the intestine, while in 7 more it involved more than two-thirds of the circumference. In at least 15 cases the mesentery as well as the intestine was involved in the laceration. Partial rupture not including all the coats of the intestine was present in 3 out of the 132 cases. Retroperitoneal rupture occurred in 4 cases. Of these, three involved the duodenum and 1 the splenic flexure of the colon.

TABLE IX.

Multiple ruptures present in 22 cases out of 132 (Berry and Giuseppe)			
"	"	20	" 134 (Hertle)
"	"	42	" 266

In 24 cases out of 132 the rupture of the intestine was complicated by the coexistence of other severe injuries. These complications were: Fracture of pelvis, 6 cases; fractures in other situations, 4 cases; rupture of liver, 4 cases; rupture of pancreas, 3 cases; rupture of stomach, 2 cases; rupture of kidney, 1 case; rupture of lung, 1 case; rupture of spleen, 1 case; of superior mesenteric and common iliac arteries, 1 each.

SYMPTOMS.

We come now to a far more important part of our subject, namely, the symptoms caused by rupture of the intestine. In 59 of our cases the notes are sufficiently full to enable us to draw conclusions with regard to the relative frequency of the more important symptoms (*see* Table X).

TABLE X—DIAGNOSTIC SYMPTOMS IN 59 CASES OF RUPTURE OF THE INTESTINES, WITH FULL NOTES.

	Present	Absent
Pain	51	—
Vomiting	43	2
Shock	28	6
Local tenderness	35	2
Rigidity	50	4
Distension	11	14
Added dullness	18	14
Rising pulse	37	6
Loss or diminution of liver dullness	6	—

It need hardly be said that if treatment is to be efficacious the diagnosis must be made early, within the first hours after the injury. We venture

to think that in many of the cases in our list the diagnosis was not made as early as it might have been. We should like especially to direct the attention of general practitioners and hospital residents, who see these cases early, to the fact that rupture of the intestine is often caused by a comparatively slight blow, and that the initial symptoms often do not suggest to the mind of the inexperienced that so serious an injury as a rupture of the intestine can have been sustained.

There is a tendency, and a very natural one on the part of a house surgeon, when face to face with a case of abdominal contusion which may possibly be one of ruptured intestine, to wait and watch for pronounced symptoms. When these latter have set in he sends for the surgeon, who thus only too often does not get the opportunity of seeing the patient until the most favourable time for operation has passed away.

In these days almost every hospital surgeon is in telephonic communication with his hospital. We venture to think that it is the duty of every house surgeon, as soon as he has satisfied himself that his patient has received a severe blow on the abdomen, to telephone forthwith to his surgeon and to give the latter the opportunity of asking further questions, and, if necessary, of at once going to see the patient himself.

Even when the blow upon the abdomen appears to have been a slight one the same course should be adopted if the patient is complaining of severe and persistent pain. This leads us to the statement that of the very early symptoms of rupture of the intestine *pain* is by far the most important. Again and again do we find it recorded that within the first few hours the patient suffered from "great" pain, "severe" pain, "intense" pain, and so forth, and that the pain was continuous. In cases where there is profound shock, often due to the presence of other injuries as well, pain may be slight, but these are not the cases in which the serious nature of the accident is likely to be overlooked (*see Case 70*).

There is yet another class of case in which pain is not necessarily a very marked feature, namely, those in which the rupture is incomplete. The pain is due to extravasation of irritating intestinal contents, and it is possible, although very unusual, for no extravasation to take place in the first few hours after the injury. It is well known that in a small rupture of the intestine the protruding mucous membrane may temporarily occlude the opening and prevent extravasation. Even in cases of complete transverse rupture there is no doubt that extravasation of intestinal contents is sometimes delayed for some hours.

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Dambrin, in his interesting monograph on "Lesions of the Intestine," quotes the theory of Jobert Lamballe, who affirms that the two cut ends of intestine retract, so as to narrow the lumen. He goes on to say that his experiments confirm this theory: "We have always seen immediately after the injury the neighbouring intestine contract over a length of several centimetres, thus obliterating the lumen; the mucous membrane usually protruded."

We believe that complete rupture of the intestine with extravasation is always accompanied by severe pain, except in certain rare cases, when the patient is profoundly collapsed or is unconscious as the result of other injuries. Whether a patient with complete rupture, but without extravasation, can be quite free from pain is, we think, open to doubt, and we hope to elicit expressions of opinion upon this point from members of the Society. There can be little or no doubt that when a patient who has ruptured his intestine remains free from pain for some little time after the injury this immunity is due to the rupture being incomplete. One or other of the intestinal coats, usually the peritoneal or submucous, has not been completely ruptured.

The following case came under the notice of one of us some years ago: A boy received a severe injury to the abdomen. He walked to the nearest hospital, but he seemed in so little pain and seemed so well that he was allowed to go home. Here he proceeded to eat a good supper, but immediately afterwards he was seized with severe pain in the abdomen and other symptoms of intestinal perforation. For these he was admitted to St. Bartholomew's Hospital, where he shortly afterwards died. At the post-mortem, performed by one of us, an extensive rupture of the duodenum was discovered. The peritoneal coat was much less torn, however, than the others, and had evidently given way secondarily.

In no fewer than 51 of the previously mentioned 59 cases it is definitely stated that pain was an early symptom, and it was usually severe. In the other 8 no mention is made of this symptom. In only 3 of the whole series of 132 cases is it definitely stated that pain was not present in the first few hours after the accident. Of these cases one (Case 70) was that of profound collapse already mentioned. Another was Case 83, in which there were two small perforations of the jejunum, and in this case the symptoms of rupture did not set in until the day after the accident; here the rupture was almost certainly a secondary one. The third case was No. 63, a remarkable case of submucous rupture of the duodenum without involvement of the peritoneum.

The specimen from this case is in the museum at St. Bartholomew's Hospital (No. 2040Ai), and by the kindness of the Curator we are able to show it to you to-night.

The patient was a boy aged 15, who was run over across the abdomen by a fire-escape. He was at once brought to the hospital in a state of collapse. He vomited several times. On the following day he seemed to have almost completely recovered, and complained only of a little abdominal pain and tenderness. Later vomiting again set in, and the boy died of intestinal obstruction on the fourth day. At the post-mortem, which was performed by one of us, a subperitoneal rupture of the duodenum was found. A clot of blood as large as a hen's egg lay between the peritoneal and the muscular coats, and completely blocked the second part of the duodenum.

A point that may be of much assistance in the diagnosis of rupture of the intestine is the shifting of the pain from one part of the abdomen to another. This is well illustrated by Case 48, under the care of one of us at the Royal Free Hospital. A man, aged 29, was struck in the epigastrium by the pole of a cart, and was brought at once to the hospital. He was suffering severe pain in the epigastrium and left hypochondrium, but was not thought by the resident medical officers to be seriously injured. Some four hours later, when seen by one of us, he was still in good condition, but his abdominal muscles were extremely rigid, he had vomited several times, and his pain, which had been continuous since admission, had now shifted from the upper to the lower part of the abdomen. A slight abrasion of the skin on the epigastrium afforded clear evidence of the exact seat of injury. The shifting of the pain downwards seemed to point to extravasation of irritating fluid, which had gravitated downwards. On the strength of these symptoms the diagnosis of ruptured intestine was made and the abdomen immediately opened. A very extensive rupture of the duodenum was found, which was sutured with great difficulty, and the patient died ten hours after the operation. The case is also interesting in that the man's general condition some three or four hours after the injury was so good that the house surgeon, in reporting the admission of the case, expressly said that he did not think that any operation was called for. He reported its admission, he said, merely because he knew that the surgeons of the Royal Free Hospital liked to be told of abdominal injuries at an early date. A somewhat similar case, in which the shifting of the seat of pain afforded material help in diagnosis, has been recorded by Mr. Battle in the *British Medical Journal*, June 13, 1908, i, p. 1412.

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Shock, as a symptom of ruptured intestine, is of very little value. It is often present, but it need scarcely be said that it is also very common in abdominal contusions of all kinds, quite apart from ruptured intestine, whereas severe and continuous pain is not. In many of our cases shock was entirely absent. Several of the patients walked home after the accident (*i.e.*, Cases 94, 100, and 113); some of them walked to the hospital (Cases 65, 82, and 110). Two of the patients went on with their work. Case 124 remained at work for four hours, and Case 131 wheeled his barrow home before coming to the hospital. Among our 59 cases it was definitely stated that shock was present in 28 cases and absent in 6.

Vomiting is an important symptom, and is generally present within the first few hours. Repeated vomiting after an abdominal contusion strongly suggests rupture of the intestine. On the other hand, vomiting may be delayed for some hours in cases of undoubted primary rupture. Case 52 was that of a man crushed between a tramcar and a wall; severe and continuous pain set in immediately, but vomiting did not begin until seven hours later. Later still he was brought to the hospital, and, when first seen by one of us, he was already in a moribund condition. The post-mortem showed a complete circular rupture of the ileum. In Cases 2, 67, 75, 100, and 123 vomiting was delayed for from two to twenty-four hours, but in all these cases pain set in immediately, and extensive, evidently primary, ruptures were found at the post-mortem or operation. Of the 59 cases, early vomiting was present in 43 cases and absent in 2. Vomiting of blood is a rare symptom, recorded in only 3 of our cases (Nos. 28, 70, and 84). All these were cases of injury to the duodenum.

Tenderness at the seat of lesion is, of course, common. Deep tenderness especially is an almost invariable accompaniment of an intestinal rupture. In our 59 cases tenderness is recorded as having been present in 35 cases and absent only in 2.

Rigidity of the abdominal muscles is of the utmost importance as a sign of severe abdominal lesion. It was present in no fewer than 50 of the 59 cases previously mentioned and absent in only 4. Hartmann states that out of 37 cases of abdominal injury that had come under his notice rigidity was present in 17. In all of these a visceral lesion was found. In the remaining 20 no rigidity was present, and the patients recovered without operation. Hertle compares the contraction of abdominal muscles in cases of visceral injury with the fixation of the hip in inflammatory disease of that joint.

A sign that may help in the diagnosis is that of added dulness,

especially local dulness at the seat of injury. Dulness due to the presence of free fluid in the abdominal cavity soon after an injury generally means blood, and is more often due to injury of the liver, spleen, or mesentery than to rupture of intestine. It may, however, be due to the escape of intestinal contents, together with peritoneal fluid effused as the result of irritation and inflammation of that membrane. A localized area of dulness in the immediate neighbourhood of the injured part is at least suggestive of rupture, and is occasionally useful as an aid to diagnosis. It is probably due partly to collapse of the intestine in immediate proximity to the rupture, partly to escape of intestinal fluids, and partly to hæmorrhage. In our 59 cases added dulness was present in 18 cases and absent in 14. Of the value of a *rising pulse-rate* in the diagnosis of ruptured intestine it is not necessary to say much, as it is well recognized by all. We would merely say that in the first few hours the pulse is often not abnormally rapid. The pulse-rate should be noted every hour in every case of suspected abdominal injury, and, if it be found to increase steadily, it affords one more reason for suspecting a septic lesion, provided that it is not due to hæmorrhage. But in many cases of rupture of the intestine the pulse-rate remains at or near the normal for several hours. The same may be said of temperature, which affords but little help in diagnosis.

We now come to a physical sign of the worthlessness of which in the diagnosis of ruptured intestine we cannot speak too strongly. And yet, if we may judge from the stress laid upon it by certain writers, and the eagerness with which it appears to be sought for by dressers, house surgeons, and others, and the faithfulness with which its absence is duly inscribed in the notes of case after case in our list, it appears to be considered by many to be a sign of much diagnostic value. We refer to the absence of liver dulness. It is true that this sign is often present in the later stages of the illness, but as a sign of rupture of the intestine in the early period—at that period at which alone operative interference can be undertaken with a reasonable prospect of success—it is, as a diagnostic sign, not only useless, but worse than useless, because deceptive. Among the 17 cases in our list in which operative interference was undertaken with success *there is not a single one in which absence of liver dulness was recorded*. On the other hand, there are several cases in which operation was not undertaken until absence of liver dulness had been noted; not one of these recovered.

Perforation of a large air-containing viscus like the stomach often permits of the escape of a large amount of gas into the peritoneal cavity. Perforation of the intestine only rarely does so. The amount of gas that

escapes from the intestine within the first few hours of its rupture is usually only trivial, not enough to show physical signs of its presence.

Melena is of no value in the diagnosis of recent rupture of the intestine. It was not present in any case in our series. On the other hand, in cases of partial rupture of the intestine, where the inner coats of the intestine alone have been torn, blood may appear in the motions, but, as a rule, not for some days. A very remarkable case came under the notice of one of us at St. Bartholomew's Hospital some years ago. A little girl of 6 fell on to a scraper, striking the upper part of the abdomen. For many days she was extremely ill, with great distension of the abdomen. She vomited blood and passed blood with her motions, but eventually made a complete recovery without any operation having been performed. There can be but little doubt that this was a case of partial rupture of the duodenum.

Emphysema of the abdominal wall was present in 1 case of our series (No. 102); the rupture was of the duodenum.

Distension of the abdomen is one of the gravest signs of ruptured intestine, but does not usually occur early. Among the 17 cases in our series that recovered after operation there was not a single one in which distension of the abdomen is stated to have been present.

DIAGNOSIS.

The diagnosis of rupture of the intestine rests not upon a single symptom or sign, but upon a careful consideration of all the facts of the case. Every case of abdominal contusion should be looked upon when first seen as a case of possible severe intestinal injury. Careful attention to the history of the accident, the manner in which the blow was inflicted, and the exact point struck may afford much help. Marked and persistent contraction of abdominal muscles, accompanied by marked local tenderness and severe pain, is sufficient to justify an immediate laparotomy in nearly all cases. The diagnosis between the rupture of the liver, spleen, or mesentery on the one hand, and rupture of the intestine (or stomach) on the other hand, rests mainly on the presence or absence of signs of hæmorrhage. After all, the important question to be decided is usually not so much, Is there a rupture of the intestine? but, Is there an abdominal lesion sufficiently grave to demand an immediate abdominal section? We venture to think that the diagnosis of an ordinary case of complete rupture of the intestine can usually be made with a tolerable degree of certainty within the first few hours of the accident. Much greater difficulty naturally occurs with those cases

in which the rupture is at first incomplete, those in which symptoms of rupture supervene many hours or even days after the infliction of the injury. In such cases as these early diagnosis often cannot be made. But all cases in which there is a suspicion that grave injury has been sustained should be most carefully watched, and when the secondary rupture does take place the symptoms will be much the same as those of primary rupture. A typical case (No. 51) may here be cited as illustration of secondary rupture. A man, aged 57, was kicked in the abdomen by a horse. When first seen soon after the accident there was slight bruising over the left iliac fossa. There were no other symptoms, and the man went home. Three hours later he returned to the hospital complaining of great pain in the abdomen; the abdominal muscles were retracted and rigid, and the pulse was 84. Death took place twenty hours after the accident, and the post-mortem showed a small rupture of the ileum. Retroperitoneal rupture is seen chiefly in connexion with the duodenum (Cases 49, 102, 121, and 132 illustrate this condition), and may be suspected when, after a severe contusion of this region, symptoms of septic absorption occur without symptoms of peritonitis.

MORTALITY.

The gross mortality in our whole series of 132 was 115. Some of the cases were suffering from other extensive injuries as well, some came to hospital when peritonitis was far advanced, a few were already moribund when admitted. Of the whole number 84 were submitted to operation. Of these 17 recovered and 67 died (*see* Table XI). Of those not operated upon, all died. Of the 47 not operated upon, 6 refused operation and 7 were considered to be too bad for operation. It must be remembered, however, that should a patient with a small or partial rupture of the intestine recover without operation the diagnosis cannot be made with any certainty, and the case will not appear in our list. Table XII shows what everyone would expect, namely, that for operations to have a fair chance of success they must be undertaken early, within the first twenty-four hours. The mortality would appear from our figures to be slightly higher in the case of operations undertaken in the first six hours. This apparent anomaly is, however, probably explained by the fact that many of the *very worst* cases, in which diagnosis presented no difficulty, would be operated upon very early. On the other hand, many of the slighter cases, in which diagnosis was at first doubtful, would not be operated upon until a few hours later. The percentage mortality in our series works out at 87.2 per cent. The

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percentage mortality of operations for ruptured intestine is usually stated to be much lower than this. Thus Hertle's series of cases shows a mortality of 76·8; Tavaststjerna's 173 cases, 76·4. But these figures, we venture to think, do not represent the true mortality, since, as we have already mentioned, they are drawn mainly from published cases. These almost necessarily contain a higher proportion of successful cases. When we come to consider the mortality at individual hospitals, a remarkable fact becomes apparent, namely, that of the 17 cases that recovered after operation no fewer than 8 occurred at one hospital, St. Thomas's. Of the 28 cases of rupture of the intestine that occurred at this hospital, 22 underwent operation and 2 were considered too

TABLE XI—RECOVERY OF 17 CASES (14 MALE, 3 FEMALE).

	Recoveries	Operation cases	Total cases
Run over	9	31	51
Blows	4	16	23
Kick	2	11	16
Crush	1	14	24
Fall	1	8	11
	17	80	125

TABLE XII—RESULT OF OPERATION IN 84 CASES (BERRY AND GIUSEPPI).

Operated on ; hours after accident	Recovered	Died	Total cases
0 to 6	5 = 27·7 per cent.	13 = 72·3 per cent.	18
7 " 12	8 = 53·3 "	7 = 46·7 "	15
13 " 24	1 = 5·0 "	19 = 95·0 "	20
24 " 28	1 = 9·0 "	10 = 91·0 "	11
After 48	—	10 = 100·0 "	10
No statement of time	2	8	10
	17	67	84

bad for operation. Eight recoveries out of 22 operations for ruptured intestine is a record of which that hospital may well be proud. It is true that in 2 of these cases the rupture was only partial, but on the other hand there were 2 cases (Nos. 108 and 116) in which death occurred so long after the operation that they might almost be considered as operative successes. We hope that if any of the St. Thomas's surgeons are here to-night, they will tell us more about the methods which in their hands have proved so successful. In the meantime we would venture to suggest that the many admirable papers that have been written upon ruptured intestine by members of the staff of St. Thomas's (Mr. Makins, Mr. Battle, and others) have duly impressed upon St. Thomas's men the importance of early diagnosis and early operation, and that this has contributed in no small measure to the success obtained. As far as we have been able to gather from the

records of St. Thomas's Hospital, their cases are seen early, diagnosed early, and operated upon forthwith.

TABLE XIII—DURATION OF LIFE AFTER ACCIDENT.

Lived		Operated upon		Not operated upon	
0 hours to 10 hours	...	3	...	4	...
11 " " 24 "	...	20	...	22	...
25 " " 3 days	...	20	...	7	...
3 days and over	...	19	...	8	...
Not stated	...	6	...	6	...
Recovered	...	17	...	—	...
		85		47	
Shortest time	...	4½ hours	...	4 hours	...
Longest time	...	26 days	...	10 days	...

Since the publication of the classical cases of Croft and Moty in 1889 and 1890, cases of recovery after operation for rupture of the intestine have become numerous, and the operation mortality has undergone marked diminution. The statistics of St. Thomas's Hospital show that it can be still further diminished. Is it too much to hope that in the next fifteen years the average mortality after operations of this class in London hospitals will be still further reduced, and not continue to stand at the comparatively high figure of 87·2?

TREATMENT.

The treatment of ruptured intestine is necessarily closely dependent upon diagnosis. If a rupture is diagnosed, or even strongly suspected, no time should be lost in opening the abdomen and dealing with the ruptured part. If the symptoms are not sufficiently clear to justify this proceeding, the patient should be most carefully watched and the surgeon be prepared to interfere directly symptoms arise. In every case of abdominal contusion the patient should, if possible, be kept at rest, and no food or drink given by the mouth until all suspicion of a serious lesion has passed away. Opium should not be given so long as the diagnosis is still uncertain. Some cases when first seen are obviously moribund, and quite unfit for any operation. The question of how far shock should lead the surgeon to defer operation is an interesting one, and worthy of discussion. Most writers advise that operative interference should be postponed until shock has passed off. We venture to think that, if the diagnosis of ruptured intestine has been made, a moderate degree of shock should not deter the surgeon from operating. We think that in such cases it is best to treat the shock actively by means of massive saline infusions, strychnine, and other suitable remedies, and then to proceed at once with the operation.

When the abdomen has been opened the rupture is usually found without any difficulty. In a considerable minority of cases, however, the rupture was not found at the operation. This happened in no fewer than 15 of the cases in our list. Of these 15, in no fewer than 7 was the duodenum the part affected. This suggests that rupture of the duodenum is not suspected as often as, according to our figures, it should be. Probably in most of these 7 cases the rupture would have been found had the duodenum been examined. It should be remembered that the duodenum should be examined both above and below the transverse mesocolon.

We are far from recommending abdominal section indiscriminately in all, or even in most, cases of abdominal contusion. It should always be remembered that in the *great majority* of cases of abdominal contusion complete recovery takes place without operation. But if a reasonable suspicion exists, founded upon the presence of such early symptoms as we have mentioned, that a serious internal lesion has been sustained, then the surgeon will do well to operate at once and not wait for further confirmation of his diagnosis.

Into the various methods of closing the rent in the intestine we do not propose to enter at any length. The main point is to effect a complete mechanical closure of the wound with as little delay as possible. Table XIV shows the various methods adopted in the 84 cases in our list. In the 17 successful cases, simple suture was employed in 15. One case recovered after the use of a Murphy's button (No. 8), and one only after excision (No. 3) (out of 12 cases). Treatment by the formation of an artificial anus was uniformly fatal. With regard to the treatment of the peritoneum, by irrigation or otherwise, by drainage or otherwise, this must to a certain extent depend upon the degree to which the peritoneum has been contaminated by intestinal contents. In our own successful case the irrigation was brief and not very thorough, in spite of the extensive effusion of intestinal contents. It is noteworthy that of 10 cases treated without irrigation no fewer than 4 recovered. With regard to drainage, out of 17 cases in which the abdomen was not drained, 7 recovered.

We should like to raise the question as to the best means of treating the terribly fatal cases of extensive rupture of the duodenum. No case of recovery after operation for rupture of the duodenum appears in our list. Indeed, we know of only one single case in surgical literature in which recovery followed an operation for ruptured duodenum. This was a case of Mr. Godwin's, of a cab driver, aged 42, who was run over. Rents of the jejunum and third part of the duodenum (the latter being 1 in. in length) were sewn up.

Mr. Moynihan's case of complete rupture at the duodeno-jejunal fold was treated by closure of the duodenum and implantation of the jejunum into the stomach. The patient recovered well from this operation, but, unfortunately, the Murphy's button which had been employed passed into the stomach and thence into the duodenum. Here it lodged, and caused a fatal perforation on the 104th day after the operation.

TABLE XIV—METHOD OF TREATMENT IN 84 OPERATIONS.

	Cases	Recoveries
Treatment of intestine :		
Simple suture	46	15
Button	14	1
Excision	12	1
Artificial anus	4	—
Treatment of peritoneum :		
Irrigation	44	10
No irrigation	10	4
Drainage after closure of intestinal wound	30	9
No drainage	17	7
Simple drainage	6	—
Rupture not found	15	—

Of the 24 cases of rupture of the duodenum in our list, in 11 no operation at all was performed; in 6 the rupture was not discovered at the operation; in 4 suture was performed; in 2 cases a Murphy's button and a Bailey's bone bobbin respectively were employed; the remaining case (No. 116) was the nearest approach to a success; it occurred at St. Thomas's Hospital. A woman, aged 30, fell on to a baluster thirty-six hours before admission. At the operation a rupture of the posterior wall of the duodenum at the junction of its first and second parts was found. Suture being found impracticable, drainage-tubes were placed down to the rupture and packed round with gauze. An abundant bile-stained intestinal discharge took place, and the patient began to emaciate. On the sixth day a second operation was performed, the duodenum being divided transversely and the ends closed. Posterior gastro-jejunostomy was performed, but the patient died some days later.

In a case (No. 48) of almost complete transverse rupture of the second part of the duodenum, operated on by one of us in 1897, approximation of the ends was effected by means of a Bailey's bone bobbin, but the patient died ten hours later. In this case the amount of extravasation of intestinal contents into the retroperitoneal tissue so soon after the infliction of the injury was very remarkable. We are of opinion that for small ruptures of the duodenum simple suture, if it can be effected, is the best means of treatment. If the rupture is too extensive to permit of this (and it often is), then the best course to pursue is probably that

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of closing the duodenum completely and performing gastro-jejunostomy. Finally, in some desperately bad cases, packing round a tube, with subsequent secondary operation, would seem to be the best line of treatment.

As regards after-treatment in our own case of recovery, the patient's abdomen was drained and he was sat up in bed from the time of the operation. This is merely in accordance with the practice adopted nowadays, we believe, by most surgeons in their operative treatment of acute peritonitis.

Of the use of massive saline injections, whether by rectum or under the skin, it is not necessary to dwell, their value being admitted by all. We think that strychnia is also useful in the after-treatment of such cases.

Another question we should like to raise is, What should be the treatment of those unfortunate patients who are not seen by the surgeon until one or two or more days have passed, and great abdominal distension has set in? We are inclined to think that in many of these cases extensive operative interference not only does no good, but actually destroys the patient's last chance of life. It must be remembered that in a certain number of cases of undoubted rupture of the intestine (especially incomplete or small ruptures) localization of extravasated material has taken place, and the patients have recovered with or without an abscess.

It is probable that certain rare cases of rupture of the intestine do recover spontaneously without operation, but it need scarcely be said that this applies only to the slighter varieties of the injury. The probability of spontaneous recovery taking place is so extremely small that it should never deter the surgeon from operation in any case which is seen by him sufficiently early.

In Table II of the Appendix will be found notes of four cases in which it was probable that a rupture of the intestine had occurred, and which ended in recovery without operation. For the first two of these cases we are indebted to our lamented friend the late Mr. Harold Barnard. Both patients were readmitted to the London Hospital four and five months after the accident on account of intestinal obstruction. At the operations then performed the evidence of previous rupture seemed fairly conclusive. The third case is one that has been published by Mr. Battle in the *St. Thomas's Hospital Reports*. In this case the ascending colon, if not actually ruptured at the time of accident, seems to have been so severely contused that it gave way secondarily. The fourth case, which was under the care of one of us, was considered to be almost certainly one of rupture of the intestine, the rupture being probably only a small one.

A perfectly healthy girl, aged 13, who had never had any symptoms of appendicitis or any other abdominal trouble, was on the evening of March 30, 1905, struck on the umbilicus by the handle of a barrow. She immediately felt sick and ill and sat down for a quarter of an hour. Her last meal had been taken four hours previously. She was nevertheless able to walk home. On the following morning she had great abdominal pain and vomited twice. She then came to the Royal Free Hospital, where she was found to have rigidity and tenderness of the abdominal muscles and pain at the umbilicus; liver dulness was normal, but her pulse-rate was 125 and the temperature 99.5°F . She went home, but returned again in the evening, and was admitted. When seen by one of us forty-eight hours after the accident she was extremely ill. The abdomen was distended, painful and tender, with marked rigidity of the muscles, especially on the left side. The liver dulness was absent; pulse 130. The diagnosis of peritonitis following a small rupture of the intestine was made. Owing to the very grave general condition, the distension of the abdomen, and the time (forty-eight hours) that had elapsed since the accident, it was thought that any operation would deprive her of her last chance of life. She was kept sitting up in bed, was given nothing by the mouth; strychnia was administered subcutaneously and saline solution *per rectum*. For three days she remained acutely ill, then slowly began to recover, and eventually left the hospital quite well.

The principal sequelæ of operations in cases that do not speedily die or recover are to be found in various inflammatory affections of the peritoneum, mainly localized abscess, and intestinal obstruction due to stricture or bands. Illustrations of the former are to be seen in Cases 108 and 122, in which subphrenic abscesses formed; and in Case 112, in which a pelvic abscess formed. The first of these died on the twenty-fifth day; the other two recovered. Case 110 affords a good illustration of intestinal obstruction following recovery from operation for ruptured intestine. The patient was a woman aged 29, who sustained two ruptures of the jejunum, closed by suture six hours after the accident. Three weeks after leaving the hospital she returned, and was again successfully operated upon for intestinal obstruction due to a band of adhesions.

The conclusions which we venture to lay before the Society are the following:—

(1) That rupture of the duodenum is by no means so unusual as is generally supposed.

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(2) That rupture of the intestine is usually produced by direct crush, and not by bursting or traction.

(3) That the early symptoms of complete primary rupture are few, but fairly characteristic.

(4) That of these the most important are severe and continuous pain, rigidity of the abdominal muscles, and vomiting.

(5) That secondary rupture is by no means uncommon, the symptoms in these cases being often delayed for hours or even days.

(6) That early operation is the only practical means of treatment.

(7) That if operation be delayed until the abdomen is distended or the liver dulness absent the patient has practically no chance of recovery.

(8) That after operation the patient should be sat up in bed and treated with massive saline infusions.

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APPENDIX—TABLES I AND II.

Table I.—132 Cases of Rupture of Intestine without External Wound, occurring in ten large London hospitals during the years 1893 to 1907 inclusive.

No.	Hospital	Name	Age	Sex	DATE OF			Nature of Injury	Symptoms and signs	Treatment	Result	Site of lesion	Condition found at operation or post-mortem
					Accident	Admission	Discharge or death						
1	Charing Cross	J. C.	31	M.	Aug. 18, 1901, 6 p.m.	Aug. 19, 1901	Aug. 19, 1901	Fell off a bicycle and was struck in the abdomen by the handle bar	Went home in a cab; severe shock; evidently in great pain; at the level of the umbilicus was a large bruise 1½ in. in diameter; abdomen moved slightly on respiration; great tenderness on palpation over the bruise; liver dullness normal; vomited several times; pulse 130; on rectal examination tenderness high up on the anterior wall	Intestine united over a Murphy's button; abdomen washed out and drained	Death 1½ hours after operation.	Small intestine	Intestinal contents in peritoneal cavity; circular tear of small intestine involving two-thirds of circumference; mucous membrane protruding
2	Charing Cross	J. C.	25	M.	April 4, 1902	April 4, 1902	April 7, 1902	Fell down and a wheel passed over lumbo-sacral spine	On admission much shock, much abdominal pain; bruises in the right lumbar region and above the right Poupert's ligament; abdomen very rigid; rallied from shock and pulse went down to 72; abdomen remained rigid and painful; vomited three times on April 6, 1902; pulse gradually rose to 92 and became feebler; temperature 97° F.; abdomen more rigid and painful, most tender in the left epigastrium; operation not thought necessary; vomited several times and became worse	April 7, laparotomy, rent 1½ hours after operation; men washed out with saline and drained; collapsed at the end of the operation; pulse 150	Death 1½ hours after operation	Small intestine	Free gas and fluid in the abdomen, no offensive smell, oblique rupture involving three-fifths circumference, mucous membrane everted, gut bruised in neighbourhood of rupture

No.	Hospital	Name	Age	Sex	DATE OF			Nature of injury	Symptoms and signs	Treatment	Result	Site of lesion	Condition found at operation or post-mortem
					Accident	Admission	Discharge or death						
3	Charing Cross	J. B.	7	M.	Oct. 3, 1907, 3 p.m.	Oct. 3, 1907	Nov. 29, 1907	Run over by a small van at 3 p.m.; wheels passed over the abdomen	On admission was crying and holding abdomen; two slight abrasions over last ribs on right side; movements slight and jerky; abdomen rigid and very tender all over; pulse 104, temperature 99° F.; 10 p.m., child sick, abdomen as before, temperature 102° F.; 10 a.m. October 4, much worse, pulse 140, temperature 102° F.; noon, face drawn, somewhat collapsed, some pain over umbilicus, abdomen markedly rigid, tender, and motionless, impairment in left flank; temperature rose to 102° F. after operation, pulse 140; vomited everything; gradually improved and left with a small sinus	Oct. 4, laparotomy 7.30 p.m., median incision, a little tumor, bid, flaky, serous fluid escaped; 2 in. of small intestine were excised, and end-to-end anastomosis performed; abdomen sponged out, drained with glass tube; pulse almost unaccountable at end of operation; infused <i>per rectum</i> , nothing retained	Recovery	Small intestine	Circular perforation in antimesenteric border, edges clean cut; some bruising of gut 1 in. to one side
4	Guy's	W. H.	42	M.	June 20, 1896	June 21, 1896	June 22, 1896	Run over	He had pain in the epigastrium and was taken home; perspired a great deal and drank 1 gallon of water; brought to the hospital on the morning of June 21, collapsed, sweating, abdomen hard and motionless, pulse 96, temperature 104° F., respiration 32; pulse gradually quickened to 122 on June 22 and temperature rose to 101° F.; bowels not opened since admission, vomited 3 pints	Brandy; refused operation	Death	Sigmoid flexure	Perforation $\frac{3}{4}$ in. in diameter in the sigmoid flexure; general peritonitis

5	Guy's	A. C.	35	M.	June 26, 1897	July 1, 1897	Run over by a van	On admission a fracture of the left side of the pelvis was detected; June 27, abdomen tense and pain all over; vomiting commenced; pulse 140; June 28, better, pulse 108, abdomen less rigid, no vomiting since 2 a.m.; June 29, abdomen still tense, looks better; June 30, abdomen less tense; July 1, abdomen much distended, fecal vomiting; bowels not opened since admission	Hot water bottles in xij. q. h.	Death	Small intestine	Several ruptures of small intestine, transverse fracture of right ilium, several fractures of left ilium
6	Guy's	J. D.	26	M.	July 24, 1898	July 25, 1898	Run over below the umbilicus	On admission, restless, pain on the right side of the abdomen; half an hour after admission became more collapsed and passed blood in his urine	Strychnine subcutaneously	Death a few hours after admission	Cecum	Rupture measuring 1 1/2 in. on the anterior aspect of the cecum
7	Guy's	W. A.	—	M.	April 20, 1899	April 22, 1899	An iron pipe fell across the abdomen	Face drawn, severe pain round the umbilicus, pulse rapid; April 21, rupture of the abdomen not distended but rigid; liver dullness normal	April 21, laparotomy, rupture sewn up, drainage	Death	Ileum	Almost complete circular rupture of the ileum
8	Guy's	G. H.	30	M.	May 23, 1899 2.45p.m.	July 27, 1899	Pole of cart struck the abdomen at the umbilicus.	Ate mackerel at 4.30 a.m.; on admission conscious; great pain in the abdomen, and knees were flexed; pulse fair; 10 minutes later became unconscious and pulse was imperceptible; brandy was injected and he revived; in the ward he was pale and collapsed; the abdomen was retracted and motionless, rigid all over and tender; liver dullness normal; pulse 100, temperature 98° F., respiration 30; dullness round the umbilicus, over an area 4 in. in diameter	Brandy; operation 2 1/2 hours after accident; venous blood in the peritoneal cavity; partial rupture of the colon sewn up; jejunum united over a Murphy's button; abdomen sponged out and then washed out with warm water; no drainage; saline infusion of 4 pints; June 4, wound gaped; June 23, wound sewn up	Recovery	Partial rupture of colon, complete transverse rupture of the jejunum	—

No.	Hospital	Name	Age	Sex	DATE OF			Nature of injury	Symptoms and signs	Treatment	Result	Site of lesion	Condition found at operation or post-mortem.
					Accident	Admission	Discharge or death						
9	Guy's	F. B.	Adult	M.	Sept. 8, 1899	Sept. 8, 1899	Sept. 9, 1899	Run over	Face pale; great pain in the abdomen; breathing irregular and almost thoracic; pulse irregular; abdomen rigid and tender. September 9, pulse rose to 142, respiration 40, temperature 97.8° F.; dulness in the flanks and hypogastrium; slight diminution of the liver dulness	Sept. 9, median laparotomy; ruptures sutured; abdomen washed out; intravenous infusion of 4 pints; brandy	Death	Ileum	Three ruptures of ileum, measuring 1½ in., 2 in. and ¾ in. in length
10	Guy's	J. M.	22	M.	Jan. 3, 1899	Jan. 4, 1899	Jan. 6, 1899	Crushed between two buffers	Stayed home for 26 hours and then came to hospital with feculent vomiting; breathing almost thoracic; dulness in left iliac fossa	Laparotomy; resection of 6 in. to 7 in. of ileum and ends united over a Murphy's button; abdomen washed out	Death	Ileum	Rupture of ileum near ileo-cæcal valve
11	Guy's	W. P.	11	M.	July 22, 1899	July 22, 1899	July 28, 1899	Run over	Pain in abdomen; muscles rigid; pulse 100	Laparotomy 1½ hours after accident; 1 ft. of small intestine resected and ends united over a Murphy's button; operation lasted 1½ hours	Death 6 hours after admission	Small intestine; partial tear of colon	Two ruptures of small intestine involving the mesentery; small tear in the transverse colon, ¾ in. to 1 in. in diameter; mucous membrane not involved
12	Guy's	R. F.	—	M.	Oct. 27, 1900	Oct. 28, 1900	Oct. 28, 1900	Received many blows on head and abdomen during a fight	Admitted day after fight, collapsed; abdomen very tense; intense pain in left side; pulse 126, breathing shallow, temperature 99° F.; vomited	—	Death	Ileum	Fractured skull; extra- and subdural hemorrhages; peritonitis; perforation of ileum 6 ft. above ileo-cæcal valve

13	Guy's	M. W.	50	M.	May 22, 1901	May 22, 1901	May 23, 1901	Block of marble weighing 15 cwt. fell on patient	On admission very collapsed; great abdominal pain (across the umbilicus); abdomen rigid but not tense; respiration quick and thoracic; pulse 120	10 hours after admission; intestine united over a Murphy's button; peritoneum washed out with normal saline; drain in pelvis	Death	Jejunum	Complete transverse rupture of jejunum and mesentery for 4 in.; blood and gas in peritoneal cavity
14	Guy's	A. C.	9	M.	Feb. 6, 1902	Feb. 6, 1902	Feb. 8, 1902	Run over; patient fell on to his face and wheel went over lower part of back	On admission pulse feeble, 104; respiration 30; vomited seven times; improved; February 7, temperature 101° F.; pulse 139; very feeble; gradually got worse	Laparotomy on Feb. 7; tear sewn up; abdomen washed out; transfused strychnine	Death	Jejunum	Perforation of jejunum 13 in. from duodeno-jejunal flexure; general peritonitis
15	Guy's	E. S.	5	M.	Dec. 24, 1902	Dec. 24, 1902	Dec. 25, 1902	Run over	On admission semi-conscious; abdomen tense and dull to the level of the umbilicus; gradually became more tense	Mother refused operation; strychnine	Death	Duodenum	Ruptured duodenum; fracture of one femur
16	Guy's	A. O.	19	F.	May 6, 1903	May 6, 1903	May 7, 1903	Run over	On admission pale; left side of abdomen was rigid, dull and swollen; respiration 28 and thoracic	Infused subcutaneously; laparotomy a few hours after admission; intestine united over a Murphy's button; strychnine; infused a second time	Death from shock	Jejunum	No faces in peritoneum; left kidney bruised; rupture of jejunum close to the duodeno-jejunal flexure
17	Guy's	J. M.	50	M.	Aug. 2, 1903	Aug. 3, 1903	Aug. 5, 1903	A youth sat on the lower part of the patient's abdomen	Abdominal pain and vomiting began soon after; on admission he was collapsed, thirsty and sweating; abdominal pain; dullness on the right side; vomiting; pulse 140	Diagnosis of ruptured liver made and operation advised, but refused	Death	Jejunum	Two small perforations, one 4 ft. from the pylorus, the other 9 ft. from the pylorus

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					Accident	Admission	Discharge or death						
18	Guy's	J. C.	49	M.	Aug. 31, 1903	Aug. 31, 1903	Aug. 31, 1903	Run over	On admission drunk and collapsed, cold sweat; pulse 60, respiration 24, temperature 93.4° F.	Refused treatment	Death	Ileum	Complete transverse rupture of ileum 4 ft. from cæcum
19	Guy's	H. W.	80	M.	1904	1904	1904	Knocked down by an engine	On admission collapsed, bloodless and clammy, did not react to stimulants; abdomen became rigid, tender and distended; died without recovering from collapse on same day	—	Death	Small intestine	Fracture of spines of lumbar vertebrae and of pelvis; rupture measuring 1 in. to 1 in. of small intestine; rupture of pancreas
20	Guy's	T. N.	27	M.	Sept. 13, 1905, 9.30 p.m.	Sept. 13, 1905	Sept. 14, 1905	Fell 50 ft. on to right side at 9.30 p.m. on Sept. 13	On admission (soon after), pale and suffering from shock; abdomen rigid, not tender; liver dullness normal, pulse 76, respiration 24, temperature 96° F.; three hours after admission abdomen became distended and dullness appeared in the left flank; extremely pale; vomited several times	Infused into the axilla; strychnine; laparotomy three hours after admission; abdomen washed out with saline; rupture not found; infused again; strychnine	Death at 10 p.m.	Jejunum	Transverse rupture 1½ in. long opposite mesenteric border of jejunum beyond duodeno-jejunal flexure; rupture of superior mesenteric artery; peritonitis
21	Guy's	A. H.	—	M.	Jan. 22, 1906	Jan. 22, 1906	Jan. 24, 1906	A weight fell on to the abdomen	On admission, lips pale; abdomen rigid, tender and motionless; pulse 120; no sickness; next day vomiting began	Strychnine transfusion; laparotomy, January 23; ruptures sutured	Death 30 hours after operation	Jejunum	Ruptures of stomach and jejunum

22	Guy's	W. T.	10	M.	Oct. 10, 1897	Oct. 10, 1897	Dec. 6, 1907	Run over by a cart	Admitted at once; colour good; in great abdominal pain; vomited blood, but nose was bleeding; same night, pulse 124, temperature 98° F., respiration 28; abdomen became tense; vomited several times; next morning abdomen rigid and tense; recovery was uninterrupted after the operation	Laparotomy 12 hours after the injury; three wounds of the intestines were sewn up; abdomen washed out and two drainage-tubes inserted above the pubis; $\frac{1}{2}$ pint of saline into the axilla during the operation; sat up in bed; continuous rectal infusion of saline; strychnine subcutaneously	Recovery	Small intestine	Three ruptures of small intestines
23	London	D.	10	M.	May 10, 1894	May 11, 1894	Received a blow on the abdomen from a barrow	On admission he was collapsed; tenderness on palpating the abdomen; no other signs; 7 a.m. next morning, abdomen became distended, very hard and tender, and frequent vomiting began; died at 9.15 a.m., 36 hours after the injury		Death	Duodenum	Rupture of the duodenum over the spine; purulent peritonitis	
24	London	W.	34	M.	Aug. 8, 1894	Aug. 9, 1894	Kicked by a horse	On admission conscious; abdomen tender and painful, especially in the hypogastrium; abdomen became distended and rigid; pulse rose to 120; vomited once		Death	Small intestine	Rupture of small intestine	
25	London	H.	21	M.	1896	1896	Hit in the back by a crane	Death in 12 hours		Death	Small intestine	Ruptured small intestine	
26	London	G. H.	14	M.	Sept. 30, 1896, 8.45 a.m.	Sept. 30, 1906	Squeezed between a lift and the floor	On admission severe shock; abdomen not rigid; tenderness in the right hypochondrium; dulness which gradually increased in the right side; vomited	Hot bottles; brandy	Death	Duodenum	Ruptured duodenum	

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					Accident	Admission	Discharge or death						
27	London	D.	10	F.	1897	1897	1897 Death in 3 days	A wall fell on the patient	On admission very collapsed; blanched; pain in the abdomen; abdomen tender and rigid on the left side; no distension; dullness on the left side; pulse 120, small and feeble; vomited two or three times	—	Death	Ileum	Complete transverse rupture of the ileum 8 ft. below the duodeno-jejunal flexure
28	London	D. M.	20	M.	1898	1898	1898	Crushed between metal plates, most of the pressure being on the epigastrium	On admission no shock; very severe pain in the epigastrium, pulse 72; vomited; became much worse; pain unbearable; abdomen tender and rigid; dullness in the hypochondrial and epigastric regions; vomited blood-stained fluid	Morphia	Death	Duodenum	Ruptured duodenum; peritonitis; bruising of stomach and both kidneys
29	London	F. E.	7	M.	July 30, 1898	July 30, 1899	July 31, 1898	Run over by a cart, which passed over upper part of the abdomen	Too collapsed for operation; no added dullness, breathing thoracic, abdomen became tender, vomiting	—	Death	Duodenum	Small rupture of descending part of the duodenum; ruptured liver; peritonitis
30	London	W. W.	48	M.	April 17, 1899	April 20, 1899	April 21, 1899	Blow on the abdomen, April 17	Came to the hospital on April 20; vomiting came on early, great abdominal pain, distension, breathlessness	Laparotomy	Death	Ileum	Rupture of ileum 6 in. from cecum
31	London	J. C.	37	M.	Jan. 23, 1900	Jan. 23, 1900	Jan. 25, 1900	Run over	On admission collapsed and drunk; impaired resonance and tenderness in the left iliac fossa; gradually became distended and tender, and liver dullness disappeared; pulse 120	No operation	Death	Sigmoid flexure	Ruptured sigmoid; peritonitis

32	London	A. T.	18	M.	1900	1900	1900	—	Died of shock	Gut resected; Murphy's button used	Death soon after	Small intestine	Ruptured intestine
33	London	J. D.	15	M.	Jan. 21, 1901	Jan. 21, 1901	Jan. 23, 1901	Fell on to a wheel	Vomited after a good meal; pulse 170; abdomen distended; hematoma of groin (right)	Laparotomy; gut resected	Death soon after	Ileum	Ruptured ileum
24	London	A. B.	45	M.	1901	1901	1901	Ladder, weight 6-7 cwt., fell on the patient	Abdomen tender and painful, vomited several times	Laparotomy	Death	Ileum	Two ruptures of ileum; general peritonitis
35	London	G. B.	17	M.	Mar. 21, 1902	Mar. 22, 1902	Mar. 23, 1902	Kicked by a horse in the epigastrium	Had much pain and vomited at once; abdomen rigid as a board all over, dull all over, pulse 120	Laparotomy; rent sutured; drainage	Death	Jejunum	Peritonitis; almost complete transverse rupture of jejunum
36	London	C. L.	12	M.	1902	1902	1902	120 lb. fell on the abdomen	On admission no collapse; pulse good but irregular; vomited three times 20 hours after admission, general peritonitis	Laparotomy; rent sutured; drainage	Death after admission	Jejunum	Tear of jejunum 1 in. long; peritonitis
37	London	V. C.	6	M.	April 6, 1903	April 6, 1903	April 10, 1903	Struck in the abdomen by a garden roller's handle	Vomited and went to sleep; on admission marked rigidity, no tenderness, absence of liver dullness, lower part of abdomen moved badly, persistent vomiting; pulse 120, small and hard; death due to exhaustion	Laparotomy 45 minutes after admission; rupture of liver found and sutured; two ruptures of stomach sutured; rupture of duodenum 1 in. from flexure sutured over a Murphy's button	Death	Duodenum	No peritonitis; death from exhaustion; button held; ruptured liver, two ruptures of stomach, rupture of duodenum (third part)
38	London	G. W.	24	M.	Mar. 31, 1903	April 3, 1903	April 7, 1903	Blow on abdomen 8 days before admission	Great pain, vomiting, and constipation since accident; abdomen distended and rigid; temperature 101° F.; peritonitis present	Laparotomy; pus in peritoneal cavity; drainage; transfusion	Death	Ileum	Ruptured ileum 7 ft. from caecum; purulent peritonitis

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					Accident	Admission	Discharge or death						
39	London	G. R.	49	M.	July 6, 1903	July 6, 1903	Aug. 27, 1903	Crushed between two vans	On admission intense pain in the abdomen; muscles of abdomen rather rigid; pulse 60, fair	Laparotomy; rent sutured; drained from two tubes in the right groin and three in the median incision	Recovery	Jejunum	Two small ruptures of jejunum near duodenum, one $\frac{1}{4}$ in. long on anterior aspect and a small puncture posteriorly
40	London	G. F.	23	M.	Jan. 25, 1904	Jan. 25, 1904	—	Crushed between the platform and a train	On admission much collapsed and blanched; after treatment with hot blankets and strychnine rectum covered, and 2 hours after admission there was pain on the left side of the abdomen, which was rigid all over; tenderness in the left iliac fossa; marked resistance in the left loin, where there was added dullness; no loss of liver dullness; vomited before the operation; pulse 108, regular and fair volume	Hot water bottles; strychnine; laparotomy; tear in jejunum sutured; irrigation; drainage; strychnine	Death	Jejunum and descending colon	At operation small tear of jejunum found; at post-mortem a second tear was found in the descending colon
41	London	E. L.	—	M.	1904	1904	1904	—	Brought in moribund	—	Death	Jejunum	Rupture 6 in. below duodeno-jejunal flexure; ruptured left kidney
42	London	A. T.	6	F.	April 12, 1904	April 13, 1904	April 15, 1904	Run over	Day after the injury sudden rapidity of pulse; abdominal rigidity; vomiting	Too bad for operation	Death	Small intestine	Rupture of small intestine and tail of pancreas; general peritonitis
43	London	A. M.	48	F.	Feb. 21, 1905	Feb. 21, 1905	Feb. 22, 1905	Run over	Abdomen tender, distended and rigid; vomited four times	No treatment	Death	Duodenum	Ruptured duodenum

44	London	—	43	M.	1906	1906	1906	—	—	Laparotomy; no tear found	Death	Sigmoid flexure	General peritonitis; tear of sigmoid flexure and mesocolon
45	London	F. B.	3	M.	1906	1906	1906	Run over	—	—	Death	Small intestine	Ruptured small intestine
46	London	J. B.	31	M.	1906	1906	1906	—	—	Morphia	Death	Small intestine	Ruptured small intestine; torn mesentery
47	Royal Free	F. H.	16	M.	Nov. 23, 1896	Nov. 23, 1896	Nov. 24, 1896	Found injured in a railway tunnel	On admission very collapsed, abdomen hard and tender, liver and spleen dullness absent; vomited two or three times	Too bad for operation	Death 4 hours after admission	Duodenum	Perforation of duodenum at junction of second and third parts, intestines bruised, left kidney bruised; blood in the peritoneal cavity
48	Royal Free	C. G.	29	M.	April 2, 1897	April 2, 1897	April 2, 1897	Crushed between the pole of a cart and another cart	Had just finished breakfast; on admission not unconscious, in great pain; pain worse in the left hypochondrium; no loss of liver dullness; pulse 68, respiration 32, temperature 95.4° F.; abdomen became tender and rigid and only moved slightly; frequent vomiting	Operation 5 hours after accident; small perforation sutured, large rupture sutured round a Bailey's tube; time, 1 hour 17 minutes; peritoneum washed out, drainage by two tubes, brandy <i>per rectum</i>	Death 10 hours after operation	Duodenum	Two ruptures, a small hole the size of a pea and a large one extending nearly round the intestine at the junction of the first and second parts of the duodenum; retroperitoneal tissues emphysematous, peritoneum congested
49	Royal Free	W. O.	11	M.	May 12, 1897	May 12, 1897	May 15, 1897	Run over, May 12, midnight	Conscious; no severe symptoms; sick twice; pulse 110, respiration 26, temperature 97° F.; at 7.30 p.m. May 13 he became unconscious; pulse 162, thready, respiration 40, temperature 102.4° F.; abdomen rigid and tender; dullness on right side continuous with liver dullness above, and extending to right linea semilunaris in front	Diagnosis of retroperitoneal rupture of the duodenum was made, for, as patient was not pale, dullness could not be due to hemorrhage; too bad for operation	Death in 3 days	Duodenum	Retroperitoneal rupture of the duodenum at junction of second and third parts, retroperitoneal tissues infiltrated with pus and intestinal contents, no peritonitis, no rupture of the right kidney

No.	Hospital	Name	Age	Sex	DATE OF			Nature of injury	Symptoms and signs	Treatment	Result	Site of lesion	Condition found at operation or post-mortem
					Accident	Admission	Discharge or death						
50	Royal Free	S. L.	50	M.	Jan. 16, 1898	Jan. 19, 1898	Jan. 20, 1898	Ran against a short post, Jan. 16, which struck him in the abdomen	Admitted January 19; general condition good, abdomen distended and tense, lower part motionless on respiration, tender; signs of free fluid in the abdomen; pulse 76, good volume, temperature 98.8° F.	Laparotomy 24 hours after admission; three perforations operation sewn up, peritoneum washed out, drainage	Death 4 hours after operation	Ileum	Three perforations of the ileum, general peritonitis
51	Royal Free	T. S.	57	M.	Sept. 8, 1898	Sept. 8, 1898	Sept. 9, 1898	Kicked in the abdomen by a horse	On admission no collapse, slight bruising over the left iliac fossa; pulse good, extremities warm. no rupture of the bladder; sent home, but returned in 3 hours complaining of great pain in the abdomen; the abdomen was retracted and moved on respiration, but not well; liver dullness present; temperature 96.6° F., pulse 84; no collapse; gradually became worse; abdomen became full and motionless, and muscles rigid; liver dullness disappeared	No operation	Death in 20 hours	Ileum	Perforation $\frac{1}{2}$ in. in diameter of ileum; edges gangrenous; peritonitis
52	Royal Free	N. E.	55	M.	June 23, 1899, 11.30 p.m.	June 24, 1899, 11 a.m.	June 24, 1899	Crushed between a tramcar and a wall at 11.30 p.m. June 23	Pain began immediately and was severe; was taken home and was very restless all night and in great agony, and vomited at 6.30 a.m.; admitted at 11 a.m. June 24; there was a large bruise over the left iliac fossa; abdomen rigid and tender, especially on left side; abdomen moved badly; liver dullness present; dullness in right flank and left iliac fossa; he was conscious and hands cold; pulse 128; abdomen became distended and liver dullness disappeared rapidly; 2 hours later pulse almost imperceptible and patient moribund	No operation	Death 24 hours after accident	Ileum	Complete circular rupture of ileum 8 ft. from ileo-caecal valve, corresponding to the bruised area of the abdominal wall; rupture of the internal and middle coats of the left common iliac artery; peritonitis

53	Royal Free	A. A.	25	M.	Sept. 26, 1899	Sept. 26, 1899	Run over by a cart	On admission looked seriously ill; was very restless and in great pain; the abdomen was retracted and rigid all over; there was a fracture of the eighth and ninth ribs on the left side, and a large lacerated skin wound above the right Poupert's ligament; liver dulness present; pulse 70, small, temperature 95.2° F.; became gradually worse and sickness began and became continuous; pulse rose to 85 in 1 hour and to 100 in 4 hours	Laparotomy 64 hours after accident; rupture intestines spunged; drainage; saline irrigation; and <i>per rectum</i> ; strychnine and morphia subcutaneously	Death	Transverse colon	Free fluid in the peritoneum; tear $\frac{1}{2}$ in. long in the transverse colon; peritonitis; duodenum bruised
54	Royal Free	—	21	M.	1899	1899	Admitted suffering from severe head injuries, received by putting his head inside the door of a swing lift, which came down and struck him on his face	Five days after admission complained of severe pain in the abdomen just to the right and above the umbilicus, where was slight discoloration; except this pain and tenderness nothing definite was discovered; abdomen moved well; no abnormal areas of dullness and no vomiting; temperature 101.6° F., pulse 132, small and irregular; diagnosis of meningitis made; rapidly became worse, delirious and restless; pain continued very severe but abdomen became only slightly distended; liver dulness normal; vomited once or twice during early part of illness and several times on day of death; considerable diarrhoea 2 days before death	—	Death 10 days after accident	Small intestine	Brain and meninges normal, no fracture of the skull, abdominal viscera adherent and covered by lymph; on right half of abdomen was an extravasation of intestinal contents which had escaped from a softened and bruised piece of small intestine 3 in. long, which had given way in several places; the holes were irregular; in right iliac fossa was foul pus; right kidney bruised

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					Accident	Admission	Discharge or death						
55	Royal Free	J. M.	16.	M.	July 13, 1901	July 13, 1901	July 14, 1901	Kicked by a horse in the abdomen	On admission too ill to answer questions; pale; abdomen hard, tender, immobile; liver dullness present; vomited very often; pulse 88, temperature 98° F., respiration 24, thoracic	Laparotomy lasting 45 minutes, 24 hours after tear sewn up. abdomen flushed out; stopped breathing during the operation	Death 45 minutes after admission	Ileum	Tear $\frac{3}{4}$ in. long, 30 in. above the ileo-caecal valve and transverse in direction; peritonitis
56	Royal Free	W. B.	51	M.	July 24, 1901	July 24, 1901	July 24, 1901	Run over, wheel passed over upper part of the abdomen, 5.30 p.m.	Admitted at once, no bruising felt so well that he wanted to go home; no abdominal rigidity at this time (7 p.m.); 8.30 p.m. worse, collapsed; liver dullness present, restless, no food since noon	Operation 9 p.m. (55 minutes); blood in peritoneal cavity; rupture sutured, saline infusion	Died 10.10 p.m.	Duodenum	Transverse tear of duodenum
57	Royal Free	C. B.	28	M.	June 4, 1902	June 4, 1902	June 5, 1902	Kicked by a horse in the left flank below costal arch, 12 noon	On admission pain, but not very severe, vomiting; pulse 56, respiration 22; some rigidity of abdominal muscles, dullness in the flanks, slight bruising; June 5, between 11 a.m. and 12 noon, pulse rapidly increased to 132, abdominal tenderness more marked and pain worse	Operation 1.40 p.m., June 5; rupturesutured	Death 5 p.m.	Jejunum	Rupture of jejunum 5 ft. from duodeno-jejunal flexure
58	Royal Free	E. B.	17	M.	July 15, 1904	July 15, 1904	July 19, 1904	Wheel of a van passed over the abdomen at the level of the umbilicus	On admission collapsed; abdomen tender, rigid, and not moving freely; pulse 66, temperature 97° F., respiration 24; rapidly became worse, abdomen more tender and signs of free fluid appeared; vomited	Laparotomy 21 hours after accident, end-to-end anastomosis; peritoneum irrigated with saline, drainage; saline subcutaneous and <i>per rectum</i> ; strychnine and morphine subcutaneously	Death 3 days after operation	Ileum	Circular rupture completely across the ileum 2 ft. above the ileo-caecal valve; tear of the mesentery; peritonitis

	W. H.	26	M.	Sept. 25, 1907	Nov. 14, 1907		See full report of case in text	Recovery	Ileum	
59	Royal Free	—				—				—
60	St. Bartholomew's	13	M.	1893	1893	Kick	—	No operation	Jejunum	Rupture of jejunum
61	St. Bartholomew's	14	M.	Dec. 21, 1894, 10.15 a.m.	Dec. 22, 1894	Run over at 10.15 a.m.	Admitted at 10.30 a.m., no collapse; pain all over the abdomen, variable dullness up to the umbilicus; continued sickness, subnormal temperature	Death in a few hours	Duodenum	An almost complete circular rupture at the junction of second and third parts of the duodenum; mainly subperitoneal
62	St. Bartholomew's	18	M.	July 6, 1894, 4.30 p.m.	July 7, 1894	Run over at 4.30 p.m.	Admitted at 4.45 p.m., somewhat collapsed; abdomen tender, rigid, and distended, no added dullness, liver dullness normal, respiration 40 and shallow; vomited at 5.30 p.m.; 8 p.m., abdomen generally tender and rigid; became delirious	Death	Duodenum	Complete circular rupture of the second part of the duodenum; peritonitis
63	St. Bartholomew's	15	M.	June 23, 1894	June 27, 1894	Run over at the level of the umbilicus	On admission collapsed; vomited once, no hematemesis, no signs of injury; next day abdominal pain began and vomiting became frequent, pulse rapid and feeble	Death	Duodenum	Submucous rupture of the second part of the duodenum; a large hematoma between the muscular and peritoneal coats had led to obstruction

No.	Hospital	Name	Age	Sex	DATE OF			Nature of injury	Symptoms and signs	Treatment	Result	Site of lesion	Condition found at operation or post-mortem
					Accident	Admission	Discharge or death						
64	St. Bartholomew's	W. S.	18	M.	April 24, 1896	April 24, 1896	April 25, 1896	Run over and brought at once to the hospital	On admission conscious and suffering from shock; pain in the abdomen and tenderness all over; slight rigidity; dulness over the hypogastrium and left iliac fossa; liver dulness normal; pulse 80; suddenly collapsed and died on April 25 (3 to 3 hours after admission)	No operation; fluids by the mouth; opium	Death	Duodeno-jejunal flexure 1 in. in length; spleen ruptured; peritonitis	Rupture at the duodeno-jejunal flexure 1 in. in length; spleen ruptured; peritonitis
65	St. Bartholomew's	J. J.	68	M.	Feb. 20, 1897	Feb. 20, 1897	Feb. 22, 1897	Run over	Walked into surgery at midnight; lower abdomen tender but not distended; vomited twice, vomit contained blood, but nose was bleeding; February 21, abdomen distended and tender, no flatus passed, and bowels not opened since yesterday; on February 22 severe abdominal pain, more distension; pulse 103, irregular, temperature 97° F.; there was present a right inguinal hernia	February 22, the hernial sac was opened and feculent fluid escaped; the abdomen was washed out and drained; only fluids by the mouth	Death	Ileum	A rent was found $\frac{1}{4}$ in. long on the convex border 6 in. from the ileo-caecal valve; purulent peritonitis
66	St. Bartholomew's	G. D.	4	M.	Aug. 23, 1897	Aug. 23, 1897	Aug. 24, 1897	Run over, wheel passed over the lower part of the abdomen	Carried to the hospital; conscious, pale and collapsed; pain in the lower part of the abdomen, which was neither rigid nor distended; pulse rapid, respiration hurried; collapse increased; pulse 150, respiration 50, temperature 98.4° F.; sick once, abdomen became tender	No operation; only fluids by the mouth; after strychnine subcutaneously	Death 13 hours after admission	Ileum	Transverse rent involving one-third circumference, 10 in. above the ileo-caecal valve; the mesentery was injured—crushed against the fifth lumbar vertebra; peritonitis

67	St. Bartholomew's	J. L.	13	M.	June 16, 1898	June 16, 1898	July 29, 1898	Run over; the wheels passed over the upper part of the abdomen	On admission collapsed; pain in the abdomen, which was retracted and moved only slightly; liver dulness present; pulse 70, small, temperature 98.4° F.; vomited once 3 hours after the accident; the abdomen soon became hard and moved less, and the pain became worse	Operation 3 hours after admission; gas and blood - stained fluid in peritoneum; rupture sewn up with Lembert sutures, abdomen flushed out with warm water and no drainage; kept under morphia; bowels opened on June 21 with castor oil, fluids only till June 23	Recovery	Small intestine	Circular rupture $\frac{1}{2}$ in. long, not involving the mesentery
68	St. Bartholomew's	J. G.	58	M.	Dec. 24, 1898	Dec. 26, 1898	Dec. 26, 1898	On December 24 he reduced a left inguinal hernia with great force	December 25, vomited several times and suffered from abdominal pain; on admission, December 26, face pallid, abdomen distended, not moving on respiration, tender and resonant; pulse 110; bowels not opened since December 24	Laparotomy Dec. 26; fluid in the peritoneal cavity; a tear was sewn up with Lembert sutures; abdomen washed out with a solution of 1 in 4,000 biniodide of mercury; no drainage	Death 2 hours after operation	Small intestine	Rupture $\frac{1}{2}$ in. long in the small intestine in the left iliac fossa
69	St. Bartholomew's	F. P.	8	M.	July 30, 1898	July 30, 1898	July 31, 1898	Run over; wheels passed below costal arch	On admission, was suffering from shock; great abdominal pain; abdomen distended; vomited frequently; pulse 160	Laparotomy July 31, peritoneal cavity contained undigested food; patient became much worse and operation was stopped and the abdomen sewn up; strychnine and morphine hypodermically; brandy per rectum	Death	Duodenum	Rupture through half the circumference of the first part of the duodenum

No.	Hospital	Name	Age	Sex	DATE OF			Nature of injury	Symptoms and signs	Treatment	Result	Site of lesion	Condition found at operation or post-mortem
					Accident	Admission	Discharge or death						
70	St. Bartholomew's	F. M.	6	M.	April 19, 1899	April 19, 1899	April 20, 1899, 3.10 a.m.	Run over at 3 p.m.	On admission, very collapsed, no pain; some bruising of the right iliac fossa, abdomen not distended, no tenderness, movements good; vomited (three or four times) blood-stained fluid; pulse weak	Saline infusion 2 pints; arms banded; legs banded; brandy	Death	Duodenum	Ruptured liver, complete circular rupture of the duodenum just beyond the pylorus
71	St. Bartholomew's	G. M.	46	M.	Sept. 21, 1899	Sept. 29, 1899	Sept. 30, 1899	Kicked by a horse in the right groin 8 days before admission	A fluctuating oedematous swelling was found on the right side above Poupart's ligament	The swelling was opened and fecal matter let out; brandy; strychnine subcutaneously	Death	Duodenum and cecum	An abscess was found occupying the whole of the right iliac fossa, and opening into the cecum on its outer wall by an aperture $\frac{3}{4}$ in. long; there was a rupture of the third part of the duodenum measuring 1 in. on its posterior wall; a sub-diaphragmatic abscess
72	St. Bartholomew's	F. R.	18	M.	Sept. 7, 1900	Sept. 7, 1900	Sept. 8, 1900	Fell off a scaffold, 25 ft. high	Admitted semi-conscious; one hour after he complained of abdominal pain; there was fullness in both flanks, abdomen moved freely; he got worse and vomiting became fecal	No operation	Death	Duodeno-jejunal flexure	Tear 1 in. long on the posterior surface of duodeno-jejunal flexure, leading into the peritoneal cavity; general peritonitis

73	St. Bartholomew's	B. E.	14	M.	June 2, 1900	June 2, 1900	June 5, 1900	Run over; the wheels passed over the lower part of the abdomen	On admission much collapsed, abdomen painful, tender, moved slightly, and slightly rigid; June 3, abdomen still painful and tender; June 4, abdomen more rigid, slightly distended, then markedly so; motionless on respiration; liver dullness normal; pulse 140; bruises round umbilicus	Laparotomy April 5, 1900. Murphys button used; no drainage; hot water bottles; strychnine subcutaneously	Death	Small intestine	Septic peritonitis, rupture of the small intestine in the left iliac fossa
74	St. Bartholomew's	R. N.	38	M.	April 10, 1901	April 10, 1901	April 12, 1901	Kicked by a horse in the right hypochondrium	On admission, pale and in great pain; dullness for 8 in. above the pubis and in the left flank; tenderness all over, especially in the right hypochondrium; urine clear; vomited; pulse 88, temperature 97.4° F., respiration 18; next day great abdominal pain, abdomen tense and rigid; little movement; no liver dullness; temperature 101° F., pulse 100, respiration 80	Laparotomy April 11; rupture closed; brandy	Death	Ileum	Peritonitis; a round perforation about $\frac{1}{2}$ in. in diameter in the ileum 2 ft. above the ileo-caecal valve
75	St. Bartholomew's	A. C.	11	M.	July 4, 1903	July 4, 1903	July 6, 1903	Run over	On admission, much pain in the abdomen; collapse; two hours after vomiting began, which became continuous; collapse became greater and patient became gradually worse; pulse 94, temperature 97° F., respiration 28	Brandy; strychnine subcutaneously; no operation	Death	Duodeno-jejunal flexure	Circular rupture at the duodeno-jejunal flexure involving three-quarters of circumference; peritonitis
76	St. Bartholomew's	F. A.	43	M.	Mar. 1, 1904	Mar. 1, 1904, 5 p.m.	Mar. 2, 1904	Knocked by a wheel on the right side	On admission collapsed; great pain in the abdomen, which was distended; vomiting; pulse 100, poor volume; temperature 97° F.	Laparotomy Mar. 1, 10 p.m.; rent sewn up; strychnine subcutaneously; brandy	Death 9 a.m.	Ileum	Free blood in peritoneal cavity; small transverse rent 3 ft. above the caecum
77	St. Bartholomew's	A. J.	5	M.	Jan. 8, 1904	Jan. 8, 1904	Jan. 9, 1904	Run over; wheels passed over the lower part of the abdomen	On admission he was suffering from shock; there were bruises over the lower part of the abdomen; abdominal pain and rigidity; later, signs of fluid appeared in the abdomen	Laparotomy, 10 p.m., Jan. 8; gut resected; anastomosis; no drainage; intravenous infusion of saline	Death from intestinal collapse	Small intestine	Fractured pelvis; sutures held at the repair in the small intestine

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78	St. Bartholomew's	W. L.	29	M.	Mar. 24, 1904	Mar. 24, 1904	Mar. 25, 1904	Run over	On admission, collapsed; great abdominal pain; bruises over the upper part of the abdomen, fewer on the left side; no added dullness; liver dullness slightly diminished; abdomen moves fairly well	Too ill for operation; died after a few hours	Death	Transverse colon and jejunum	Fracture of pelvis, complete circular rupture of transverse colon close to the hepatic flexure, complete circular tear of jejunum 6 in. beyond the duodeno-jejunal flexure
79	St. Bartholomew's	W. C.	22	M.	Oct. 20, 1906	Oct. 20, 1906	Oct. 24, 1906	Struck by the pole of an omnibus in the abdomen	On admission, semi-conscious, pain in the epigastrium, continuous in character; there was a bruise at the umbilicus and abdominal tenderness; vomiting; pulse increased from 88 to 116	No operation	Death	Jejunum	Complete lacerated transverse tear of the jejunum, 4½ ft. from the duodenum
80	St. Bartholomew's	L. W.	35	F.	Mar. 26, 1906	Mar. 28, 1906	April 1, 1906	Fell down stairs	Vomited once after taking castor oil; on admission, abdomen was distended, rigid, and tender; doubtful whether free fluid was present or not; pulse 104, respiration 28	General peritonitis; no operation	Death	Ileum	General peritonitis; rupture just above the ileo-caecal valve for three-fourths of circumference
81	St. Bartholomew's	A. B.	47	M.	Nov. 17, 1906	Nov. 17, 1906	Nov. 19, 1906	Run over	On admission, abdomen was rigid and tender, liver dullness normal; there were fractures of the seventh and tenth ribs on the right side; pulse 80, regular; lower third of the abdomen became motionless; expression anxious; Nov. 18, severe abdominal pain; fecal material appeared through the tubes	Rupture of liver diagnosed; laparotomy Nov. 18, no gas or faeces seen, abdomen irrigated and drained in each loin, liver sutured	Death	Jejunum	Liver torn, small intestine torn completely across 4 ft. from the duodenum, mesentery slightly torn; general peritonitis

82	St. Bartholomew's	L. S.	39	M.	Feb. 21, 1907	Feb. 24, 1907	Feb. 25, 1907	Kicked by a horse 60 hours before admission	No localization; he was able to walk; pain increased, no vomiting, bowels not opened; for last 30 hours hiccough; bowels not opened after many rectal injections; abdomen distended, moves badly, rigid and tender all over; movable dullness in both flanks, liver dullness decreased	Exploratory laparotomy, Feb. 24, rupture sewn up, peritoneum washed out	Death	Jejunum	Rupture of jejunum size of a saucer; general peritonitis
83	St. Bartholomew's	W. N.	26	M.	Dec. 13, 1907	Dec. 13, 1907	Dec. 14, 1907	Dragged by a horse 7 ft. on to a pole, which struck abdomen in the region of the gall bladder	No marks seen on the abdomen; no pain; passed a restless night; abdominal symptoms appeared out of a next day and patient became much worse	No operation	Death	Jejunum	Peritonitis; two small perforations 5 ft. from the duodeno-jejunal flexure
84	St. Bartholomew's	C. D.	27	M.	June 1, 1907	June 1, 1907	June 2, 1907	Fell 7 ft. on to a pole, which struck abdomen in the region of the gall bladder	Admitted 6.15 p.m. collapsed; abdomen rigid and tender over the right hypochondrium; liver dullness normal, no dullness in the flanks; vomited; pulse 76, temperature 97.4° F.; 8.20 p.m., vomited blood; pulse 104, temperature 100° F.; 11 a.m., liver dullness diminished, pulse 132	Operation 18 hours after admission; peritoneum contained bile-stained fluid; rupture sewn up; washed out with saline	Death 8 hours after operation	Duodenum	Rupture of duodenum 1 in. beyond the pylorus
85	St. Bartholomew's	G. C.	—	M.	May 16, 1907	May 16, 1907	May 17, 1907	Squeezed between buffers	Severe shock; intense abdominal pain; intense thirst; pulse 84, fair volume, temperature below 95° F.; pulse became very quick and irregular	—	Death	Transverse colon	Complete transverse rupture of the transverse colon 15 in. from caecum; pancreas contused
86	St. Bartholomew's	C. E.	19	M.	June 7, 1907	June 7, 1907	July 30, 1907	Run over	Collapsed; severe abdominal pain; vomiting; admitted 11.30 a.m., pain all over the abdomen; vomited several times; recti very rigid; 9 p.m., temperature rose to 102° F., pulse 108; liver dullness normal	Operation about 14 hours after accident, median laparotomy; tear sutured; drainage; saline per rectum; abdomen irrigated with normal saline	Recovery	Jejunum	Tear of jejunum, size about a shilling, close to the duodenum and near the mesenteric border; tear of mesentery; intestines injected

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					Accident	Admission	Discharge or death						
87	St. George's	—	58	M.	Mar. 31, 1898	Mar. 31, 1898	—	Struck by 3 cwt. of lead (which he was carrying on head) on lower part of left side of abdomen of a woman	He had a left inguinal hernia for 12 years; rupture disappeared after blow, excessive pain immediately; on admission, excessive pain and collapse, bruising of lower part of abdomen; diagnosis of ruptured intestine made, but operation refused until pain and collapse increased; after operation much pain, pulse 102; vomited seven times	Laparotomy 5 hours after admission; the sac of an acute vaginal hydrocele, which had not been noticed, was excised; hernial sac opened and bile-stained fluid found; abdomen opened, rupture sutured, abdomen washed out and drained; hernial sac removed, operation lasted 1½ hours; condition fair after operation; nutrient enemata till April 5	Recovery	Small intestine	Zig-zag rupture measuring ¾ in. on free edge of small intestine
88	St. George's	—	17	F.	May 20, 1898	May 22, 1898	May 30, 1898	Run over by a cab	Much collapsed, abdomen bruised, rigid and tender, considerable abdominal pain, vomiting	Immediate laparotomy, rents sutured, abdomen cleansed; no drainage	Death from shock	Duodenum and ascending colon	Rent of serous and muscular coats of ascending colon, rupture of second part of duodenum about size of sixpence, head of pancreas smashed

89	St. George's	J. P.	55	M.	Dec. 2, 1900	Dec. 2, 1900	Dec. 4, 1900	<p>On admission very collapsed; in great pain; abdominal muscles rigid; no distension; vomited once; sweating profusely; pulse small and weak; temperature 96° F.; December 8, vomiting continued; bowels not opened; abdomen distended and absolutely rigid; hepatic and splenic dullness absent; temperature subnormal; December 4, very ill; vomiting continuous; pulse 120; very weak; temperature 96.8° F.</p>	<p>December 2, refused consent to operation; December 4, median laparotomy; free gas and feculent fluid in the peritoneal cavity; rest excised and united with Lembert sutures; free irrigation with boracic lotion; drainage; strychnine, ether and morphia subcutaneously; brandy</p>	Death	Jejunum A	<p>large rent in the jejunum 6 in. below the duodenum</p>
90	St. George's	F. M.	35	M.	Oct. 31, 1902	Oct. 31, 1902	Oct. 31, 1902	<p>Crushed by a lift</p> <p>Admitted collapsed, almost pulseless; conscious; great abdominal pain; lower ribs on left side fractured</p>	<p>Laparotomy one hour after admission; blood and faeces in peritoneal cavity; colon united over a Murphy's button; peritoneum irrigated; brandy transfused to 8 pints; marked temporary improvement; drainage</p>	Death 14 hours after operation	Transverse colon	<p>Complete transverse rupture of colon at its centre; separation of symphysis pubis and sacro-iliac synchondroses</p>

No.	Hospital Name	Age	Sex	Date of			Nature of injury	Symptoms and signs	Treatment	Result	Site of lesion	Condition found at operation or post-mortem
				Accident	Admission	Discharge or death						
91	St. George's	F. O. 46	M.	Mar. 3, 1903	Mar. 3, 1903	Mar. 4, 1903	Knocked down by a dray	On admission no collapse; large hematoma of abdominal wall above and to left of umbilicus; abdomen moves well; no tenderness or fluid in the flanks; temperature 97° F., pulse 90; March 4, vomited much; pulse rose to 130 in the evening; abdomen soft and moving well; 9.30 p.m., pulse 150, very feeble; diagnosis of internal hemorrhage	Operation 16 hours after accident; laparotomy; blood and fecal material in the peritoneal cavity; no gas; portion of gut excised and gut united over a Murphy's button with Lembert sutures; bruised ovum excised; drainage above pubis and in right loin; peritoneum irrigated; transfused during the operation	Death 3 hours after operation	Jejunum	Loop of jejunum torn through in two places, leaving an isolated piece $\frac{3}{4}$ in. long
92	St. George's	G. W. 18	M.	June 1, 1903	June 1, 1903	June 2, 1903	Run over, 7 p.m.	Had tea and sandwiches 5 p.m.; not collapsed, very thirsty; abrasion at the level of the umbilicus, pain and tenderness along the upper third of the right rectus, which was rigid, movements restricted and painful; no vomiting; June 2, 12.30 p.m., suddenly worse, pulse 140	Parotom y. June 2, 3 p.m., right rectus torn, meso-ecum and mesentery of ascending colon bruised, bowel greenish yellow, no rupture found, no gas or blood in peritoneum; too ill for further interference	Death, 11.45 p.m.	Jejunum	Acute peritonitis, feculent fluid in peritoneum 26 in. from duodenum, a longitudinal rent $1\frac{1}{2}$ in. long opposite the mesentery; mesentery torn 5 ft. lower down

93	St. George's	J. D.	50	M.	Aug. 21, 1903	Aug. 21, 1903	Crushed between two tons of coal and a wall, 1.30 p.m.	Very collapsed, pain over the abdomen, which was rather rigid and moved badly, tenderness in right iliac fossa; pulse 82, temperature 97.6° F.; 3.30 p.m., very collapsed; pulse 60, wiry, temperature 96.2° F., abdomen distended, very rigid, liver dullness present	Laparotomy Aug. 21, 8 p.m., blood in peritoneal cavity, tear in liver sewn up; torn duodenum sutured (three stitches); strychnine subcutaneously	Death, 1.30 a.m.	Duodenum	Liver ruptured, duodenum torn across in the third part and extensively lacerated
94	St. George's	B. D.	20	M.	Jan. 27, 1904	Feb. 2, 1904	Struck by a piece of wood in the abdomen to right of navel	Pain not very bad; left off work and went home, and as pain became worse went to bed; vomited and sweated; admitted 32 hours after accident; there was a painful area 1½ in. below and to right of umbilicus, slight rigidity and distension, no added dullness, pulse full and good; January 29, fairly comfortable; January 30, sickness, bowels not opened since accident, abdomen soft; January 31, abdomen rigid, very restless, frequent vomiting; February 1, bowels not opened, pulse 130; worse	Operation, Jan. 28, free gas in peritoneal cavity, a perforation found and sewn up with Lembert sutures; all intestines eviscerated and washed with saline; drained; transfusion 2 pints; strychnine subcutaneously	Death	Jejunum	Perforation size of a threepenny piece with ragged edges; peritonitis
95	St. Thomas'	—	60	M.	1894	1894	Fell from a cart; the previous admission	Frequent vomiting since; abdomen distended and tender and very rigid; bowels not opened, good result after an enema; abdomen became distended again; great pain in the right iliac fossa; temperature 101.4° F.	—	Death 4 days after admission	Sigmoid flexure	General septic peritonitis; minute hole in sigmoid; no sign of ulceration
96	St. Thomas'	W. T.	57	M.	1895	1895	Run over by a cart; the wheels passed over the left inguinal region	Site of wheels marked by a graze; tenderness in inguinal region and left thigh; abdomen flaccid; slight pain; next morning nothing important found; in the afternoon became worse; abdomen distended and rigid; dullness in the left flank, not movable; features pinched; vomiting	Laparotomy 20 hours after accident, rent sutured; great collapse	Death a few hours after operation, 24 hours	Ileum	General peritonitis; rupture of ileum 1 ft. from pylorus; rupture measures ¾ in. in the long axis of the bowel and on the free surface, edges ragged

No.	Hospital	Name	Age	Sex	Date of			Nature of injury	Symptoms and signs	Treatment	Result	Site of lesion	Condition found at operation or post-mortem
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97	St. Thomas'	T. Y.	12	M.	1895	1895	1895	Run over by a cart day before admission	No symptoms until morning of admission; some collapse, anxious expression; severe pain in lower abdomen, above the pubis; no dullness in the flank; no retention, constant vomiting	Laparotomy 24 hours after injury; blood in peritoneal cavity; resection of ruptured gut and end-to-end anastomosis	Death in 36 hours after accident	Ileum	Peritonitis; transverse rupture of ileum through half circumference, 4.4 in. from ilio-caecal valve, edges fairly cleanly divided
98	St. Thomas'	—	24	M.	1895	1895	1895	Kicked in the abdomen	Bruise below and to left of the umbilicus; collapsed; abdomen hard, distended and not moving well; dullness over front of abdomen; vomiting began soon after the accident; after a few days, vomiting began again and patient became collapsed	Laparotomy 5 hours after admission; 13 in. of small intestine excised and laterally anastomosed; end-to-end anastomosis of a third rupture with a Senn's plate; abdomen washed out and drained; saline infusion at second operation; sutures had given way; an artificial anus was made	Death 5 hours after operation	Jejunum	Three ruptures of jejunum, first 20 in. from pylorus, upper two complete transverse ruptures 8 in. apart, lower one not extending up to the mesentery; peritonitis
99	St. Thomas'	W. S.	38	M.	1896	1896	1896	Kicked by a horse 2 hours before admission	Admitted shortly after accident, unconscious; no collapse and no bruising; tender spot internal to right anterior superior spine; impaired resonance in the left flank; general condition good; liver dullness present; vomiting began in a few hours, then shifting dullness appeared in the flanks	Operation 21 hours after accident; sero-pus evacuated; rent sewn up with 3 rows of Lambert sutures; abdomen sponged out and closed; no drainage; great shock	Death 3 hours after operation	Jejunum	Rent involving one-third circumference, which started at the mesenteric border; transverse in direction, 8 ft. from the pylorus; general septic peritonitis

100	St. Thomas'	J. G.	52	M.	1897	1897	1897	Three days before admission Immediately pain in the abdomen; went home and took castor oil next day but with no effect; vomited 34 hours after accident; severe pain until admission; abdomen rigid, distended and generally tender; no dullness; condition fair	Laparotomy third day after second accident; two day perforations after sutured; fecal material in peritoneal cavity; abdomen irrigated, drained; vomiting continued	Death second day	Ileum	General peritonitis; upper rent measured 1 in. and was transverse, and 9 in. from caecum; lower rent 1 in. long and transverse and 6 in. from caecum
101	St. Thomas'	J. W.	49	M.	1897	1897	1897	Pole of van struck left side and pinned him to a wall three days before admission Little shock; great pain in the left iliac fossa; tenderness and dullness in that region; vomited; given morphia; pain and tenderness relieved; on third day pain returned and vomiting started; temperature 101° F.; pulse strong; restless; became collapsed; pulse feeble and running; fourth day, vomiting and abdomen distended; liver dullness diminished	No operation	Death on fourth day	Jejunum	General peritonitis; rupture $\frac{1}{4}$ in. in diameter on free border 5 ft. from duodenum
102	St. Thomas'	E. D.	19	M.	1897	1897	1897	Two wheels passed over abdomen 2 hours before admission In state of shock; restless; pulse feeble; no vomiting or shifting dullness; emphysema of abdominal wall, and liver dullness absent; distension; severe pain	Laparotomy 3 hours after in a few accident; mesentery empty; 12 hours shock during operation; no rent found	Death 12 hours	Duodenum	Emphysema of abdominal wall; rent on posterior non-peritoneal surface of duodenum $2\frac{1}{2}$ in. from pylorus and $\frac{1}{2}$ in. long; gas in retro-peritoneal tissues, then to a rent in anterior abdominal wall and so to peritoneal cavity

No.	Hospital	Name	Age	Sex	DATE OF			Nature of injury	Symptoms and signs	Treatment	Result	Site of lesion	Condition found at operation or post-mortem
					Accident	Admission	Discharge or death						
103	St. Thomas	E. C.	37	M.	1898	1898	1898	Kicked by a horse in the abdomen 45 minutes before admission	Doubled up and sweated profusely on admission, 3 hour after accident; pain midway between pubis and the umbilicus; no shock; abdomen tender over an area the size of the palm of the hand above the symphysis and towards the left iliac fossa; here the note is less resonant and the muscles rigid; vomited; temperature 99.6° F.; 3½ hours after accident, great abdominal pain, restlessness, and difficulty of respiration; vomited twice; area of tenderness greater and note over it duller	Laparotomy 7 hours after accident; semi-digested food thirty in peritoneal cavity; rent closed by Lembert sutures in a transverse direction; abdomen sponged out; no drainage; fed by mouth on the 3rd day; discharged on the 37th day	Recovery; left on 30th day	Small intestine	Ecchymoses of mesentery; perforation of small intestine on the free border admitting tip of finger; mucosa slightly prolapsed; coils of collapsed gut corresponding to area of dullness
104	St. Thomas	F. N.	26	M.	1898	1898	1898	Struck by a piece of wood in the right iliac fossa 45 minutes before admission	Vomited 10 minutes after accident; admitted 3 hour after accident; in pain, still vomiting, sweating; breathing shallow and rapid, legs drawn up, pulse 88, temperature 99° F.; grazed above right Poutart's ligament; abdomen tender and rigid, especially in the right iliac fossa; no loss of liver dulness and no added dulness; pulse gradually quickened, abdomen more rigid and tender; progressed satisfactorily	Laparotomy 8 hours after accident; free left on fluid with fecal fortieth odour in peritoneal cavity; rents closed, abdomen irrigated with disinfected water; gauze drain down to caecum; no shock after operation	Recovery; left on 30th day	Caecum	Operative findings: Omentum in situ, bruised, a longitudinal rupture measuring 1½ in. and involving peritoneal coat of caecum; a second rupture, transverse in direction, at the junction of the caecum and colon, forming a valvular opening into caecum; retro-peritoneal hematoma of right iliac fossa and emptying upwards for 3 in. along outer border of colon

106	St. Thomas	G. H. 62	M.	1898	1898	1898	<p>Knocked down and kicked by a horse in the abdomen 2 hours before admission</p> <p>Severe abdominal pain, abdomen moving and not tender, no added dulness; no vomiting; restlessness; pulse rapid; severe shock</p>	Died in a few hours	Death less than 24 hours after accident	Small intestine	Bruise of transverse colon which reached near pelvis; a rupture of small intestine 8 in. long near the mesentery
106	St. Thomas	W. H. 41	M.	1898	1898	1898	<p>Caught between two buffers 2 hours before admission</p> <p>Slight abrasion in right iliac fossa; pain across the lower abdomen; slight tenderness at the abrasion; abdomen moves well, no dulness in the flank, no shock or vomiting; pulse good, temperature 98° F.; second day, evening, abdomen became tender but not distended; no free gas in the abdomen; vomited before death; pulse became more rapid and feeble</p>	No operation justifiable on second day	Death in 48 hours	Ileum	Rupture of the size of a three-penny piece on the free border of the ileum
107	St. Thomas	F. K. 7	M.	1898	1898	1898	<p>Fell 8 ft. 4 hours before admission</p> <p>Compound fissured fracture of frontal bone; transverse bruise at the level of the anterior superior spines; abdomen moved well except lower part; liver dulness normal; pulse 120, temperature 97.4° F.; next day abdomen became more tender and rigid, and there was dulness in the right iliac fossa; slight pain in abdomen</p>	Laparotomy 26 hours after accident; rupture sutured; irrigation; drainage; morphia; digitalis and brandy	Death after operation	Ileum	General peritonitis; rupture 6 in. from cecum; mucosa prolapsed and almost closing aperture

No.	Hospital	Name	Age	Sex	Date of		Nature of injury	Symptoms and signs	Treatment	Result	Site of lesion	Condition found at operation or post-mortem
					Accident	Admission						
108	St. Thomas'	W. B.	27	M.	April 4, 1898	April 30, 1898	Wheel of cart passed over the abdomen below the umbilicus 2 hours before admission	On admission pain in the abdomen; a few hours later began to be sick, and sickness continued for 9 hours; pulse fairly good; pain increased and abdomen became tender and rigid; no loss of liver dulness; no added dulness; temperature 100.6° F.; vomiting continued in spite of morphia; April 8, vomiting ceased, but hiccough commenced; April 10, pain in the back; April 12, stitches removed; April 14, wound gaped; intestines which protruded were washed and wound sewn up; April 22, temperature irregular, reaching 102.4° F.; dulness below spine of right scapula; breath sounds and vocal vibrations absent; April 26, aspirated, nothing found; patient became worse	Twenty hours after accident median laparotomy; sero-purulent material escaped; the small intestines were turned out; tear closed with Lambert sutures; peritonitis removed; irrigated with hot distilled water; drainage; nutrient enemata, morphia; April 28, right pleura opened but nothing found; the abdomen was then opened and adhesions felt, but no abscess found	Death on median laparotomy; sero-purulent material escaped; the small intestines were turned out; tear closed with Lambert sutures; peritonitis removed; irrigated with hot distilled water; drainage; nutrient enemata, morphia; April 28, right pleura opened but nothing found; the abdomen was then opened and adhesions felt, but no abscess found	Ileum	At operation a longitudinal tear, $\frac{3}{8}$ in. long, 12 in. from ileocecal valve; mucous membrane protruded at post-mortem; $\frac{1}{2}$ of pus in the pocket of peritoneum, behind right lobe of the liver; a few adhesions of the intestines
109	St. Thomas'	H. E.	26	M.	1899	1899	Caught in the epigastrium between the pole of one van and the tailboard of another van	Collapsed, pain and bruising in the epigastrium; abdomen moves well, liver dulness normal, no shifting dulness; vomited several times; first day, bowels opened, temperature 99° F., pulse 84; second day, abdomen tender, vomiting less; third day, marked diskension	Operation on fourth day after 5 hours injury, no free fluid, no ruptures found	Death after operation	Duodenum	Local peritonitis over the upper part of the abdomen, three rents of the third part of the duodenum

110	St. Thomas	M. R.	29	F.	1899	1899	1899	Run over	Walked in 1 hour after accident: slight abdominal pain and tenderness, abdomen moves well, liver dullness normal, pulse 86; 3 hours later, severe abdominal pain, abdomen rigid and moving badly, slight dullness in the left flank, vomiting began, pulse 120; 12 days after operation abdomen became rigid and very tender, pulse 160, temperature 100° F.; improved and was discharged; 24 days later returned with partial obstruction due to a band between two loops; band divided; she became distended, and at a third operation, 24 hours after second operation, multiple punctures of the small and large intestines were found; abdomen was irrigated with normal saline and closed, magnesium sulphate 3j every 2 hours was given, and patient made an uninterrupted recovery	Median laparotomy 6 hours after accident, home sutures, abdomen washed out with distilled water, no drainage; at second operation, 12 days later, a small hole was found 1 in. below the other two in the centre of an area from which the peritoneum was stripped; there was free gas in the peritoneal cavity, the opening was sutured, abdomen irrigated and drained	Recovery: went home on sixtieth day after accident	Jejunum	Two holes, size of one shilling piece, 4 in. below commencement of jejunum, one at the mesenteric attachment, the other opposite; mucous membrane was prolapsed
111	St. Thomas	W. S.	4	M.	1900	1900	1900	Fell down some steps 4 days before admission	Abdominal pain and constipation; castor oil was given, and this was followed by stercoraceous vomiting and abdominal distension	Bowel resected, and end-to-end anastomosis performed; normal saline into both axillae	Death 24 hours after operation	Small intestines	Adhesions were found between two coils, and on separating them the rupture was found
112	St. Thomas	J. H.	10	F.	1901	1901	1901	Run over by a cab	On admission abdomen painful and very tender, rigid, especially on the right side, breathing almost thoracic, vomiting, anxious look, pulse 120; on 10th day after operation abdomen became distended and rigid and vomiting began; pulse 100	Operation, incision through right rectus, rupture sutured, peritoneum washed out with saline; no drainage; saline and brandy per rectum; on 13th day after first operation a pelvic abscess was opened and drained	Recovery: went home on sixtieth day	Jejunum	No free fluid; lower ileum bruised, peritoneum stripped off in two places; there was a transverse rent $\frac{3}{4}$ in. long in jejunum; the mucous membrane was prolapsed

No.	Hospital	Name	Age	Sex	DATE OF			Nature of injury	Symptoms and signs	Treatment	Result	Site of lesion	Condition found at operation or post-mortem
					Accident	Admission	Discharge or death						
113	St. Thomas	W. N.	8	M.	1901	1901	1901	Kicked by a horse	Walked home, became collapsed the same evening, took castor oil; next morning worse; on admission 21 hours after accident, abdomen distended and dull in flanks	Too collapsed for operation	Death 1 hour after admission	Ileum	Acute general peritonitis; perforation $\frac{1}{2}$ in. long in the lower part of ileum; some mesenteric hemorrhage
114	St. Thomas	F. H.	11	M.	1902	1902	1902	Run over by a two-wheeled vehicle	Transverse bruise just above the umbilicus; some shock; pulse 100, temperature 98.6° F.; no free gas or fluid; next day abdomen rigid; vomiting; pulse 120, temperature 100.2° F.; suffered from broncho-pneumonia, otherwise recovery uneventful	Laparotomy through right rectus; large hematoma below the right fascia ilaca; peritoneal and muscular coat torn over the hepatic flexure, a few sutures used; peritoneum washed out with normal saline; no drainage	Recovery; went home on twenty-sixth day	Hepatic flexure of colon	Lumen not opened
115	St. Thomas	P. J.	33	M.	1904	1904	1904	Ran while drunk against an iron railing, which struck patient in the mid-abdomen	Suffered great pain and vomited a great deal; admitted 17 hours after; abdomen rigid, distended and tender; pulse 80, temperature 100° F.; 5 hours later liver dullness absent; pulse 120	Median laparotomy; rupture sutured; abdomen washed out; became collapsed; saline infusion	Death a few minutes after operation	Ileum	A perforation, size of a lead-pencil, in the upper part of the ileum; gas in the peritoneal cavity; general peritonitis

116	St. Thomas	M. W.	30	F.	1905	1905	1905	Fell on to a baluster 36 hours before admission	Struck right side of abdomen; severe pain in right hypochondrium; abdomen rigid, especially the right rectus; much shock; pulse 136, temperature 98° F., respiration 36; condition good for some days; abundant discharge containing bile and faeces; Sixth day, pulse 112, temperature 97° F., respiration 24; seventh day, pulse 120	Laparotomy through right rectus; peritonitis near duodenum; suture impossible; drainage-tubes down to ruptures; second operation, sixth day, duodenum divided transversely and ends sutured; posterior gastro-enterostomy	Death ninth day	Duodenum	Acute peritonitis, rupture of duodenum at junction of first and second parts on posterior wall, measuring 1 in. long
117	St. Thomas	W. E.	26	M.	Mar. 16, 1906	Mar. 16, 1906	April 2, 1906	Two wheels passed from left to right across lumbar and umbilical regions	Vomited soon after; abdomen rigid, not much distended, moving fairly on respiration; tender over the left rectus; liver dullness diminished; bruise over the right iliac fossa; pulse 100, temperature 99° F., anxious expression	Operation, March 17, 7 hours after accident; no gas; peritoneal coat of caecum sewn up; no drainage	Recovery	Caecum	Peritoneal coat of caecum torn; hæmorrhage into the mesentery of the small intestine
118	St. Thomas	R. P.	52	M.	April 17, 1906	April 17, 1906	April 18, 1906	Squeezed between the shaft of a wagon and a wall	On admission persistent shock; upper half of right rectus rigid; tenderness in region of umbilicus; some dullness in right flank; movements not good; sick three times; pulse 68, temperature 97° F.; watched for a few hours; vomiting continued; rigidity increased; abdomen became somewhat distended, and dullness increased	Laparotomy 14 hours after accident; drainage; no rupture found; condition too serious to allow continuation of operation	Death after operation	Duodenum	No peritonitis; hæmorrhage into mesentery and into transverse mesocolon; tissues round right kidney partially digested; rupture of duodenum 5 in. from pylorus involving two-thirds of circumference

No. Hospital	Name	Age	Sex	Date of			Nature of injury	Symptoms and signs	Treatment	Result	Site of lesion	Condition found at operation or post-mortem
				Accident	Admission	Discharge or death						
119	St. Thomas'	14	M.	Oct. 4, 1906	Oct. 4, 1906	Oct. 8, 1906	Fell on to a post	Admitted 4 hours after; great abdominal pain and tenderness; some rigidity; no area of added dullness; liver dullness present; vomited several times; pulse 100; October 5, sick once; no increase of rigidity; no pain; pulse 120, temperature 103° F.; liver dullness gradually diminished	Laparotomy 29 hours after accident; rent sutured; abdomen freely washed out; no drainage	Death 48 hours after operation	Jejunum	General peritonitis; a small recent perforation 4 ft. from the duodeno-jejunal flexure, several bruises lower down
120	St. Thomas'	61	M.	Oct. 26, 1907	Oct. 26, 1907	Oct. 27, 1907	Knocked down by a tram	Pulse rapid and poor; liver dullness disappeared; shifting dullness in the flanks; shock	Refused operation; strychnine and hot water bottles	Death in a few hours	Duodenum	Blood in the peritoneal cavity; subperitoneal hemorrhage; at the junction of the stomach and duodenum was a rent 2 in. long extending into the retroperitoneal tissues; lower margin of the gut was intact
121	St. Thomas'	33	F.	July 14, 1907	July 14, 1907, 12.15 a.m.	Sept. 6, 1907	Run over by a horsed omnibus; wheels passed from left to right at the level of the lower ribs	Collapsed condition; no bruising; constant and severe abdominal pain on left side, where was tenderness, rigidity over left hypochondrium; pulse good; during night complained of pain and vomited once; 10 a.m., pulse 100, respiration 32, pain on left side, rigidity on left side, marked tenderness along line of descending colon, fecal fistula until August 31	Laparotomy 10 hours after accident; small rupture leading into peritoneal cavity, with no extravasation of feces; sutured, transverse incision then made outwards, and peritoneum raised and rupture sutured; drainage; first incision sewn up; anti-colic serum	Recovery	Splenic flexure of colon	Extra-peritoneal rupture of splenic flexure of colon, complete and measuring 1 in.; gas in mesocolon; small opening leading into the peritoneal cavity

122	St. Thomas'	G. E.	28	M.	Oct. 15, 1907	Oct. 15, 1907	—	Fell 30 or 40 ft. from a crane	Became unconscious and was brought to the hospital shortly after; severe abdominal pain, not decreasing; abdomen moves badly; dullness in the left flank (not movable), liver dullness normal; pulse 88, respiration 32, temperature 97.4° F., pulse increased to 104, dry pleurisy right lung, left-sided basal pneumonia on October 30; dull on right side up to middle of scapula. November 9; temperature fell after operation, and rose again with similar dullness by December 14, improved since; temperature normal for three weeks; not yet discharged, January 15, 1908	No operation	Death	Jejunum	General peritonitis; transverse rupture, $\frac{3}{4}$ in. long, opposite to mesenteric border, 3 in. below duodeno-jejunal flexure; fracture of sternum at junction of body and ensiform cartilage	Complete rupture of small intestine, longitudinal in direction and measuring 14 in.; omentum bruised, mesentery torn at site of intestinal rupture
123	University College	W. M.	37	M.	May 7, 1895	May 7, 1895	May 8, 1895	Run over	On admission was suffering from shock; severe abdominal pain over upper part; May 8, pain still severe and in same position; abdomen moves well, no distension or added dullness; respiration 40 and shallow; vomited three times, and after a time vomiting became constant; abdominal facies; rapidly became worse; cold; pulse thready; abdomen moving to end	No operation	Death	Jejunum	General peritonitis; transverse rupture, $\frac{3}{4}$ in. long, opposite to mesenteric border, 3 in. below duodeno-jejunal flexure; fracture of sternum at junction of body and ensiform cartilage	Complete rupture of small intestine, longitudinal in direction and measuring 14 in.; omentum bruised, mesentery torn at site of intestinal rupture
124	University College	D. F.	26	M.	July 9, 1895	July 9, 1895	July 10, 1895	Fell 40 ft. on to back at 6.30 p.m.	On admission was pale and drowsy, and suffered from pain in the lower part of the back, slight pain in the epigastrium; vomiting; abdomen rigid but not distended, very tender, and moved slightly with respiration; no added dullness; liver dullness normal; pulse 100, good	Laparotomy 21 hours after accident; rupture sewn up; drainage; abdomen flushed out; ether hypodermically; brandy <i>per rectum</i>	Death	Jejunum	Fracture of twelfth dorsal vertebra; circular rupture, 4 in. long, 15 in. below duodenum; mucous membrane pouling; intestine congested	Complete rupture of small intestine, longitudinal in direction and measuring 14 in.; omentum bruised, mesentery torn at site of intestinal rupture

No.	Hospital	Name	Age	Sex	DATE OF			Nature of injury	Symptoms and signs	Treatment	Result	Site of lesion	Condition found at operation or post-mortem
					Accident	Admission	Discharge or death						
125	University College	E. G.	68	M.	Oct. 5, 1900	Oct. 5, 1900	Oct. 7, 1900	Run over	On admission slight bruising round the umbilicus; abdomen very painful and distended; October 6, pain over lower part of abdomen; abdomen still distended, no added dullness; liver dullness normal; abdomen moves on respiration; temperature 101.6° F.	Morphia	Death	Ileum	Peritonitis; small rupture just above ileocecal valve, closed by the mesentery of another coil being adherent; pelvis fractured
126	University College	A. H.	20	M.	Aug. 17, 1900	Aug. 17, 1900	Aug. 18, 1900	The end of a plank struck patient full in the middle abdomen	In great pain, was sick once on way to hospital; abdomen rigid; no added dullness; vomited frequently; shifting dullness developed in lower part of abdomen; pulse became much worse, liver dullness normal	Laparotomy; gas and blood in peritoneal cavity, artificial anus made; transfused; died 1 hour later	Death in 12 hours	Jejunum	Small intestine torn three-fourths extent of circumference 9 in. from duodenum
127	King's College	E. R.	38	M.	1898	1898	1898	—	No notes	Enterectomy and anastomosis by Mayo Robson's hobbin; no drainage; sponged out	Death 3 days after accident	Small intestine	Four ruptures of small intestine; compound fracture of the right leg
128	King's College	—	23	M.	1900	1900	1900	—	No notes	Operation 8 hours after admission	Death 24 hours after accident	Jejunum	Ruptured jejunum
129	King's College	—	51	M.	1901	1901	1901	Front part of a cart struck him across the abdomen	Went on with his work for 4 hours and then came to the hospital looking very ill; up to this time no vomiting, but this symptom soon began; no bruises, severe pain and tenderness in the lower part of the right side, abdomen became more rigid but not distended, pulse increased in rapidity	Laparotomy 98 hours after injury; an artificial anus was formed with the aid of a Paul's tube as patient's condition was bad, saline was used to wash out the peritoneal cavity, continuous infusion	Death 5 hours after operation	Ileum	No gas noticed, but intestinal contents were free in peritoneal cavity; large rupture in the lower part of the ileum

130 King's College	M. R. 16	M. Dec. 27, 1901	Dec. 27, 1901	Run over, Dec. 28, 1901 1 p.m.	Very collapsed, but conscious; abdomen rigid, urine normal; 3.30 p.m., abdomen becoming distended and tender, emphysema of right side of chest developed, pulse rapid and feeble; no vomiting	ab-L a p a r o t o m y 3 hours after accident, no rupture found, abdomen washed out with saline	Death 18 hours after operation	Ileum	Two circular ruptures of the upper part of the ileum, lower lobe of right lung ruptured, no ribs fractured
131 Middlesex	W. A. 63	M. Nov. 10, 1899	Nov. 10, 1899	Struck in abdomen by the handle of a barrow which he wheeled and which was run into by a cart, 1.30 p.m.	He wheeled barrow home, and had no pain for some time; pain then began and became severe; on admission severe pain, abdomen quite rigid, much tenderness left iliac fossa, liver dullness present; pulse 96, temperature 99° F., urine contained a little blood; bronchitis for a few days, abdomen tender and distended for a few days	Laparotomy 8 hours after accident; bladder uninjured, intestinal rupture found just below tender spot, rupture sewn up with Lembert sutures; sponged; no drainage	Recovery	Small intestine	Rupture 2 in. long of small intestine
132 Middlesex	W. H. Adult	M. June 25, 1907	June 25, 1907	Crushed between the tailboard of a cart and a wall	On admission abdomen moved little on respiration, considerable tenderness over right rectus, over which a hematoma could be felt; liver dullness present, no added dullness, no sickness; pulse 90, temperature 98.6° F.; during night sick several times; June 26, pulse 120, temperature 100.2° F., sickness continued, abdomen rigid, liver dullness present; 3 p.m., pulse 140, temperature 101° F.; abdomen very rigid and tender; vomit fecal, dullness in both iliac fossae; 5 p.m., pulse 160, condition otherwise same; never rallied	Laparotomy 7.30 p.m., right of middle line, no perforation found; abdomen sponged out, drainage from both flanks and from above pubis and below ensiform cartilage	Died 6 a.m.	Duodenum	Right rectus ruptured, pelvis full of bile-stained fluid; no gas; intestines covered with lymph, mesentery along its attachment to the posterior abdominal wall undergoing fat necrosis; a rupture of the third part of the duodenum behind the mesentery

Table II.—Cases of Probable Rupture of Intestine recovering without operation.

No.	Hospital	Name	Age	Sex	DATE OF		Nature of injury	Symptoms and signs	Treatment	Result	Site of lesion	Condition found at operation or post-mortem
					Accident	Admission						
A	London	—	3	F.	—	—	Run over; wheel passed over abdomen, Aug. 3, 1905	Profoundly collapsed but recovered; readmitted December, 1905, with complete intestinal obstruction of 8 days' duration	Ileum found strictured and adherent to abdominal wall in two places about 2 in. apart; large aperture in mesentery; intestines ruptured on separation and Paul's tube inserted	Died	Ileum	—
B	London	—	40	M.	—	—	Kicked in the pit of the stomach	Laid up several days; vomited; abdominal pain ever since; 3 months later admitted suffering from colic and contracting coils of intestine	A loop of jejunum was found kinked, with scar crossing obliquely, and covered by adherent omentum; lateral anastomosis	Re-covered	Jejunum	—
C	St. Thomas'	P. S.	24	M.	Aug. 1, 1895	Sept. 3, 1895	Caught between two wagons passing each other and turned round until they came to a standstill	Only superficial contusions found at hospital; was kept in bed at home; had slight abdominal pain, no sickness or other symptoms; temperature 100° F. in evening; August 26, got up and was seized with severe pain; no vomiting; swelling on right side extending from costal arch to right Poupart's ligament; this was large, tense, elastic, dull on percussion and fluctuating in lower half; sent to hospital in great pain; urine contained blood	Sept. 6, abscess opened and drained; no definite opening into intestine found for three days; feces escaped	Re-covered	Ascending colon	(?) severe contusions and secondary slough

D	Royal Free	L. F. 13	F. Mar. 30, April 1, 1905	May 6, 1905	<p>30, Previously perfectly healthy; after the accident she sat down for 15 minutes and felt ill, but walked home; last meal at 5 p.m.; March 31, much pain; vomited twice; bowels opened; 2 p.m., came to the hospital; abdomen moving well on respiration and not noticeably distended; some rigidity and tenderness of abdominal muscles; pain round umbilicus; no added dullness, and liver dullness normal; pulse 125, good; temperature 99.5° F.; she went home; 8.30 p.m., returned with more pain; admitted to hospital; rigidity and tenderness increased; pulse 130; abdomen distended, moves badly, left half markedly rigid; liver dullness absent; pain worst in left lumbar region; April 3, vomited twice; pulse rose to 144, became gradually better; April 14, slight bulge of anterior wall of rectum; April 17, tenderness of both iliac fossae present; left quite well</p>	<p>Complete rest in bed; no food covered by mouth; saline <i>per rectum</i>; strychnine; diagnosis: almost certainly rupture of some part of the intestine; but as 48 hours had elapsed since accident and the abdomen was greatly distended it was thought preferable not to operate</p>	Rupture of some portion of the intestine
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DISCUSSION.

The PRESIDENT (Mr. Warrington Haward) said he was sure everyone would agree that the Section had had the privilege of listening to a most interesting paper, prepared with Mr. Berry's usual care and industry. Any contribution towards the early diagnosis of abdominal injuries must be most valuable to all surgeons, and the clear manner in which the authors had pointed out the symptoms which were chiefly to be depended on for the diagnosis of rupture of the intestine would be remembered by them all as a most useful addition to knowledge. He hoped surgeons present would relate any similar experiences which they might have had.

Mr. G. R. TURNER desired to relate a case which was not included in the present paper. It was that of a boy in whom the rectum was ruptured without any external wound. He was not able to say exactly how it was ruptured, as the history was very confused. The boy was said to have been jumping over a broomstick, which passed up his anus and into the abdominal cavity, causing rupture. When admitted into St. George's Hospital he had the ordinary symptoms of collapse—vomiting, and a very rigid lower abdomen, the upper part, however, remaining quite supple. The case was diagnosed as a rupture of the rectum, and this could just be felt on examining the lower bowel in the usual way. He did a laparotomy, and found faecal matter in the pelvis and abdominal cavity. He removed this and stitched up the wound in the rectum, which was in the upper part, as best he could, passing the stitches through all the coats of the bowel as rapidly as possible. He afterwards packed the parts with cyanide gauze, but did not use any drain. He treated the injury to the bowel as one would treat an abscess of the appendix. He thought it in this case inadvisable to do any washing out. The boy made a good recovery, and his bowels were opened naturally on the fifth or sixth day; they were not stimulated to do so by means of purgatives or enemata. Subsequent examination of the rectum, as far as it could be felt, showed that there was nothing in the nature of a stricture, and he believed the patient was still alive and well; he saw him some months after the operation, but not very recently. He believed the success of the treatment was due to the fact that the case was seen early, and operation was undertaken within six hours of the injury. He had also been lucky enough to have another successful case, which was included in the present table, and was said to have taken one and quarter hours for the anæsthetic administration and operation. It was a case of hernia, in which a weight fell on the rupture, and the diagnosis of ruptured bowel was made and operation was urged on the patient; but it was not until three or four hours afterwards, when his increasing pain compelled him, that consent was given. He cut down first on the hernial sac, and found also tense hydrocele. In addition to having eventually to open the abdomen and suture the bowel, he dealt with the hernia

and the hydrocele. He removed the sac of the hernia and did a radical cure. The patient never gave any anxiety at all. He was not fed by the mouth for five days, and the bowels were left to act naturally. The authors advocated putting the patients in the sitting posture; Mr. Turner thought the important point was to keep them in one position, not allowing them to turn or twist about. That was of great consequence in all cases where there had been an extravasation of abdominal contents or where an abscess had been dealt with. The President and he some thirty years ago saw a case of the late Mr. Pollock of ruptured duodenum long before it became the fashion to open the abdomen. The patient lived for some five days without taking anything by the mouth. When it was thought to be safe to feed in this way he was given a little milk, and only then did the acute peritonitis supervene which carried him off. That case made a very great impression on his mind as showing the necessity of starvation in cases of suspected rupture of bowel.

Mr. S. G. KIRKBY-GOMES said the following case came under his own observation. When he was surgeon-in-charge of a Government general hospital in British Borneo, a Chinaman who had been thrown off a trolley was brought in. He hit his shoulder against a rail, and had an extensive bruise on his right side. The house surgeon detected a fracture of the right humerus and dislocation of the shoulder on the same side. As the man was in great pain he was given opium, though it was against the rules to permit opium smoking by Chinamen in hospital except under very exceptional circumstances. He vomited once in the hospital. Mr. Kirkby-Gomes came to the hospital four hours after the admission of the patient and found the latter moribund. There was increased dulness over the right hypochondrium, and the spleen was much enlarged. A ruptured liver was suspected. He died an hour after Mr. Kirkby-Gomes got to the hospital. At a post-mortem examination, made for medico-legal purposes, he discovered a ruptured duodenum. He mentioned the case to emphasize the danger of giving opium in such cases and also for its interest in the fact that the diseased spleen (ague-cake) was not ruptured, as it was apt to mislead the surgeon and put him off the correct diagnosis in cases of ruptured intestine.

Mr. PERCY SARGENT said he thought few could be found who would quarrel with Mr. Berry's admirable summary of the main points in such cases. He agreed that absence of liver dulness was of no practical value, and he would be inclined to place rather more emphasis than the authors had done on the pulse-rate. A steadily increasing pulse was of the very greatest value, and in some of the cases he had seen it had been practically the only symptom pointing to the necessity for operation. In one case in particular—one of bullet wound in the abdomen, near the umbilicus—there were no symptoms of peritonitis beyond the gradually accelerated pulse-rate; there was no pain, rigidity, vomiting, or dulness. The pulse-rate gradually went up from 90 to 120, and on that symptom alone the abdomen was opened, and several perforations were found in the small intestine. With regard to the rather striking difference in

the mortality figures at St. Thomas's as compared with other hospitals, he suggested that it might be due to the fact that there was a resident surgeon on the spot, which, until quite recently, had not been the case at other hospitals. This circumstance would conduce to the earlier recognition of the injury and operation, either by the surgeon-in-charge or by the resident surgeon, at the earliest possible moment. In all cases of peritonitis no doubt the date of operation was the prime factor.

Mr. GIUSEPPI, in reply, said the authors were much indebted to Mr. Turner for his remarks. They did not include the first case he mentioned as it was regarded as a case of perforation through the rectum. There were records of several other cases at London hospitals of the same kind, principally from falls on spikes. In one, the rectum was perforated by the sigmoidoscope, and that also was regarded as a case of perforation.

Mr. BERRY, in reply, said he would like to corroborate the view that opium should not be given so long as the diagnosis was doubtful, and to mention a case which occurred at the Royal Free Hospital some years ago. The house surgeon had correctly diagnosed a rupture of the intestine, and at once telephoned for his surgeon. Then he gave the patient opium to relieve the pain, and when the surgeon arrived the man seemed so extremely well that operative measures were postponed for several hours, and much valuable time was thereby lost. He hoped Mr. Sargent did not think the authors undervalued the importance of a rising pulse-rate, but they had perhaps said less about it than they might have done because it was already so well known. The symptom upon which they particularly wished to lay stress was pain, the importance of which as a diagnostic sign was perhaps not sufficiently recognized. The authors were much obliged to those who had discussed the paper.

Surgical Section.

November 10, 1908.

Mr. J. WARRINGTON HAWARD, President of the Section, in the Chair.

Why and How the Surgeon should attempt to Preserve the Appendix Vermiformis—its Value in the Surgical Treatment of Constipation. With a Series of Cases briefly reported.

By C. B. KEETLEY, F.R.C.S.

IN this paper I wish to report my experience of appendicostomy, appendicotomy, and appendix transplantation, and to deal with the subject in the following order: After an historical introduction will be stated the uses of these operations, next their advantages and justifications under various circumstances, as well as their drawbacks, and how some at least of these can be minimized. Then the selection of cases and the technique of the operations will be dealt with, and finally the cases will be classified, reported, and commented on.

The first appendicostomy was performed by Dr. Robert F. Weir, of New York, and published in the *New York Medical Record*, 1902, lxii, p. 201. Within two or three years the operation was repeated by other surgeons. [For references, see my paper in the *British Medical Journal*, 1905, ii, p. 863.] All these early cases were of mucous, membranous, ulcerative, syphilitic, or amœbic colitis.

In the spring of 1905 Dr. Seymour Taylor passed over to me for surgical treatment a case of inveterate constipation, and on March 10 of that year, operating on this patient, I finished an exploratory laparotomy by performing appendicostomy. The result was very satisfactory, and the case was demonstrated to many colleagues and post-graduates. Three months afterwards Mr. R. W. Murray published a letter in the

British Medical Journal suggesting a temporary appendicostomy for the purpose of washing out the bowel in cases of chronic constipation.¹

This led me to publish my case in the *Journal* of the following week (June 17, p. 1358), and I received from Mr. Murray an amiable congratulatory letter, to which I replied, recognizing his priority in the point of publication in the Press. But the new edition of one of our best works on "Surgery" is unintentionally unjust to me in saying that Mr. Murray suggested the procedure before I practised it. I had operated on my case three months before Mr. Murray's letter.

In the spring of 1905, after reducing by laparotomy an intussusception in a child at the West London Hospital, I performed appendicostomy with the threefold object of (1) anchoring the cæcum to prevent recurrence of the intussusception, (2) affording a superior means of administering injections to prevent collapse, and (3) to permit the interior of the injured intestine to be laved with hot water. This and other cases were reported in the *British Medical Journal* for October 7, 1905, in a paper entitled "Appendicostomy," which I had read at the annual meeting of the Association in July. In this paper I recalled the fact that ten years before, at the Medical Society of London, I had suggested that the appendix should be used as a channel, instead of cæcal colotomy, in the treatment of intestinal obstruction. I also mentioned a case in which I had recently done this operation, *i.e.*, appendicostomy, for the escape of fæces as distinguished from the admission of injections; and at the same time I pointed out the possible value of appendicostomy in the treatment of typhoid.

In the *Lancet* for February 17, 1906, Sir William Bennett published a thoughtful and interesting lecture in which he discussed among other questions the possible usefulness of appendicostomy—(1) in the treatment of "a certain form of intestinal distension occurring after abdominal operations in very toxic cases, and in connexion with some acute diseases, *e.g.*, pneumonia," and also as a safety valve after some operations of intestinal resection, and (2) as "a means of administering nourishment, and an alternative to the rectum for that purpose." The latter was one of the purposes for which I had used it, as reported in my first paper.

My experience of appendicostomy for these purposes will be related, with reports of the cases, in this paper.

In the *Lancet* for April 14, 1906, I published a paper, described by its

¹ *Brit. Med. Journ.*, 1905, i, p. 1299.

title, "On Appendicostomy and on Appendicæcotomy¹ as a substitute for Cæcal Colotomy; Appendicostomy and Enterostomy in the Treatment of Typhoid Fever," in which I related additional cases of successful use of the appendix as a spout for giving egress to fæces, and in which I gave my views on the subject of the surgical treatment of typhoid fever.

In the interval between this paper and my first on "Appendicostomy," Dr. William Ewart had been extremely anxious to utilize that operation in the treatment of typhoid fever, and had found on the cadaver little or no difficulty in introducing an instrument through the ileo-cæcal valve by way of the appendix. I repeated Dr. Ewart's experiment in one of my appendicostomy cases, and found the proceeding quite practicable and harmless; but this was not a case of typhoid.

On June 21, 1907, in the Cavendish Lecture,² I returned to the subject of the surgical treatment of constipation, and at the same time raised the question of substituting, in suitable cases, for appendicectomy the preservation of the appendix and its transplantation into the abdominal wall, after correcting such faults of the organ as might be found, *e.g.*, by removing concretions, undoing kinks and twists, dilating strictures, &c.

Of course, during this period of several years, the literature of appendicostomy has received other contributions. But they have, so far as I have observed, consisted of cases and series of cases of colitis and dysentery, or of casual expressions of opinion. *An exception is an excellent thesis on the subject by Dr. Aurelio Stresino, of Genoa.*

In a paper based on a rather limited experience of appendicostomy in the treatment of dysentery in the Philippine Islands, it was condemned for its liability to lead to gangrene of the appendix. I hope to show how that complication can be avoided. Again, a most able surgical friend of mine remarked that he would rather have his appendix in his pocket than transplanted into his abdominal wall. If he ever had his appendix in his pocket, his experience of that kind must have been confined to a single specimen.

Mr. Seton Pringle, of Dublin, in the *Medical Press and Circular* for November 28, 1906, described an ingenious mode of utilizing appendicostomy as a means of obtaining direct access to the small intestine. This was by anastomosing the distal end of the appendix with the ileum and opening the appendix in the middle. He performed this

¹ An operation permitting the appendicostomy to be extended by notching the cæcum when an unusually large opening is required.

² *Lancet*, 1907, i, p. 1761, and *West Lond. Med. Journ.*, 1907, xii, p. 171.

operation successfully and gave a very clear account of the technique, with diagrams.

Thus the uses of appendicostomy practised or suggested up to date may be tabulated as follows, for—

(1) Colitis of various kinds—muco-membranous, ulcerative, amœbic, syphilitic, tuberculous, &c.

(2) Certain forms of intussusception (to prevent recurrence, &c.).

(3) Intestinal hæmorrhage.

(4) Typhoid fever.

(5) Cases of enterectomy and colectomy (as a safety valve).

(6) Intestinal distension in toxic conditions.

(7) The administration of nutrient enemata *per appendicem*; and for

(8) Constipation.

And, further, there is appendicostomy as part of the technique in the conservative practice of transplanting the whole or the greater part of the appendix into the abdominal wall instead of removing it by an appendicectomy.

I especially desire to show in this paper and by the case reports attached (1) that transplantation of the appendix vermiformis, so that the whole or the greater part of it from its root in the cæcum lies permanently embedded in the abdominal wall, will produce the good results of excising it; (2) that it is a practicable and safe operation; and (3) that transplantation of the appendix should in many cases be preferred to appendicectomy. Afterwards an attempt will be made to prove that when constipation requires to be treated surgically, appendicostomy should be the operation chosen. Then will be given the inferences to be drawn from my cases as to the value of appendicostomy for various purposes. Finally, abstracts, short but not so brief as to be worthless, will be given of all my cases of appendicostomy and of appendix transplantation up to date (October, 1908), and, as throwing some light on them, there will be added cases of cæcostomy, in which that operation had to be substituted because the appendix had previously been removed in one case and was impervious in the other.

Appendicitis is a dangerous disease, not because of the nature of the appendix, but because of its position. The dangerous and even seriously troublesome results are due to its relation to the peritoneal cavity. This is not disputed. How trivial a malady even perforative appendicitis becomes when the appendix is securely embedded in the substance of the abdominal wall is indicated by the history of Cases 5 and 6. In the more interesting of these, two fish-bones, each

1 in. long, lodged in the appendix. The patient was a lady aged 78, in whom, two years previously, the appendix had been transplanted and dilated to permit the egress of fæces during an attack of acute obstruction of the large intestine, and then kept open to permit daily injections of hot water for the prevention of constipation. The fish-bones caused a small abscess, or phlegmon, beneath the cicatrix, which in two or three days opened and allowed them to escape at once. Compare this with the probable course had the appendix been lying loose in the peritoneal cavity.

In Case 32 the appendix was transplanted while still acutely inflamed. It was stiff, swollen, highly injected, and surrounded by recent sticky adhesions. This inflammation appeared to continue until, forty-eight hours after the transplantation, its apex was opened, a stricture near its base dilated with a rubber catheter, and the appendix and cæcum washed through with warm water. All pain disappeared at once, and convalescence went on without interruption. There are other cases in this series, some of them less striking, perhaps, but similar in kind. In one instance, in which the appendix was transplanted successfully, that organ was (1) kinked, (2) twisted, (3) strictured, and (4) it contained two small concretions. All these evil conditions were removed by simple measures and have shown no signs of recurrence. In another, the symptoms had been caused by a large concretion (1 in. long) which threatened perforation near the middle of the appendix. It was pressed out through a small longitudinal incision, the latter closed by a single Lembert's suture of fine catgut, the appendix transplanted and its apex opened at once as a safety valve to prevent extravasation through the lateral incision. The inference which I draw, therefore, from experience is that an appendix transplanted is an appendix disarmed.

We are then faced with the questions, Is the appendix any good? Is it worth preserving? Sir William Macewen demonstrated that the appendix has physiological uses, possibly, if not probably, of considerable importance, and that it is not the useless, merely vestigial organ it has been represented. But it has also a potential surgical value which appears greater and more astonishing the more experience one gains of appendicostomy, and the clearer one's insight becomes into a class of abdominal and other troubles which, among adults of various ages and especially of the female sex, are exceedingly common.

I have already at least twice been unable to attempt appendicostomy in cases in which that operation was indicated, because the appendix had been previously removed. In one of these cases the excised appendix had been almost quite healthy.

So far I have been dealing with facts. But there is one hypothesis of such far-reaching importance, and put forward by a biologist so eminent, that it must not be ignored, namely Professor Metchnikoff's belief that the degenerations of old age are to a large extent the effects of toxins manufactured by the bacteria which swarm in the large intestine. Among the facts bearing on the question he has reminded us of the great ages attained by birds, such as parrots and ravens, which have no large intestine. For this and other reasons, considerable portions of the human colon have been excised, a comparatively serious procedure at the best, and one of obviously partial if not doubtful efficacy in this connexion because sufficient large intestine must be left for the purpose of absorbing the water required by the blood.

Appendicostomy, on the other hand, when used for regular, frequent, and considerable injections of water, at one and the same time feeds the blood with liquid and washes out of the large intestine its noxious germs and their toxins.

I do not pretend that it has yet been proved that appendicostomy will stave off or postpone the degeneracy of old age; what I urge is that, in the view of such a striking possibility, we should preserve appendices instead of sacrificing them, *i.e.*, in all favourable cases transplant them instead of amputating them. An appendix transplanted into the abdominal wall need not be used for an appendicostomy at the time; but it is there if ever it should be wanted.

THE SELECTION OF CASES SUITABLE FOR TRANSPLANTATION.

Further experience has confirmed my belief in the rules I gave in the Cavendish Lecture.¹ Obeying these scrupulously, a large number of appendices which come to operation through appendicitis are more suitable for transplantation than for excision.

¹ Op. cit.: "What cases are suited for this parietal transplantation of the appendix?—(1) the healthy appendix in some cases of colitis, and in some of doubtful nature; (2) appendices of which a fair length of the proximal part is free from stricture, kink, ulceration, and perforation, or which can be opened towards the distal end and cured of their defects. The following cases are more or less unsuited: (1) obliterated appendices; (2) tuberculous, actinomycotic, and cancerous appendices; (3) appendices gangrenous or perforated near the proximal end; (4) appendices of which the base cannot be brought up to the parietal peritoneum without undue tension; (5) those of which the meso-appendix is not long enough to permit them to be sufficiently straightened out or to be brought into the abdominal wall without dangerously interfering with their blood-supply, but this may be ignored if the appendix is not opened too early; (6) appendices which cannot be placed in good position in the abdominal wall without interfering with such drainage as the case may require. An extremely thick and fat meso-appendix is unfavourable, but not an absolute contraindication."

TECHNIQUE.

The first steps in the operation are not different from those employed for appendicectomy, but the upper end of the parietal incision should not be too low or too near to the middle line, or there will be an increased possibility of the base of the appendix not coming up to the wound without tension. Appendix transplantation is a plastic operation, and one of the first rules in plastic cases is to avoid tension. An appendix and a cæcum which do not readily and easily come to the surface when first exposed can often be persuaded to do so by patience and a little gentle traction, and especially if abnormal adhesions or needless external and posterior peritoneal connexions be separated or if the parietal incision be extended upwards and outwards. Take care of the free margin of the meso-appendix, which contains the main appendical artery.

If the appendix is kinked, a little patient stretching will sometimes straighten it out; if twisted it may be untwisted; but do not squeeze it too tightly or seriously bruise it.

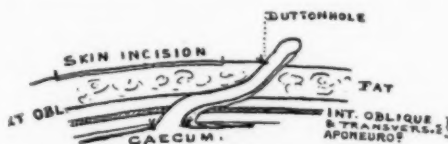


FIG. 1.

The muscular layers are placed in this diagram rather wide apart to prevent confusion. This exaggerates the S curve of the transplanted appendix.

Small or movable concretions may be pressed towards one end or the other of the organ, large ones squeezed out through a short longitudinal incision, which can be cleansed and closed again with a Lembert's suture of fine catgut. In such cases the distal end of the appendix should be opened as soon as the operation is concluded.

The appendix should be laid in an easy position obliquely in the abdominal wall, by preference with its apex upwards and outwards. Its base should lie beneath the middle or upper third of the wound, and frequently a special button-hole be made through the skin for the apex of the appendix to go through and a passage burrowed beneath the fat between the original wound and this button-hole (fig. 1).

The cæcum itself close to the base of the appendix should be fixed to the edge of the peritoneum either by three or more interrupted sutures

or by a simple continuous suture. If the latter is used care must be taken not to strangulate the appendix, and the suture should be carried through the meso-appendix, not round the appendical artery.

If the meso-appendix is thick or wide it may be button-holed and the peritoneal edges stitched together through it. One stitch may suffice for this. The rest of the peritoneal wound should now be closed with a continuous suture. Then suture the transversalis and the obliquus externus successively in such a way that more of the appendix lies beneath the latter than beneath the former, as in the diagram. Then with a very fine catgut suture, not drawn tightly, persuade the fat to cover all the rest of the appendix except 1 in. of the distal end, which should project beyond the skin and be united to the skin by a single silkworm-gut suture. I nearly always use continuous sutures for every layer—peritoneum, deep muscle, external oblique, fat, and skin—employing silkworm-gut for the skin, moderately strong catgut for the muscles and aponeuroses, and very fine catgut for the fat.

As a rule I think it is better not to open the appendix for forty-eight hours. By that time it is adherent in its new place and the skin wound is united. Therefore, should any accidental pollution be caused by the opening, it will not affect directly the surface of the appendix where it is about to obtain a new source of nutrition through its adhesions in the abdominal wall. In this way gangrene is prevented. Another rule important for the prevention of gangrene is not to leave a catheter in the appendix. When it is absolutely necessary to leave one in it should be so small as not to distend the appendix, because tension, as is well known, throws a strain on nutrition.

To prevent gangrene, therefore—(1) do not open the appendix until it has adhered to its bed; (2) do not leave a catheter in unless absolutely necessary; (3) if it is necessary, leave a very small catheter in; (4) take every care to keep the wound aseptic and unpolluted. This, as well as the avoidance of tension, is the object of rules 1, 2, and 3.

The appendix is quite insensitive and needs no anæsthetic. It can therefore be dealt with in several stages. It is generally best opened by simply cutting it nearly in two, thus:—



FIG. 2.

Then with a pair of forceps on the distal lip of the notch, and after hæmostasis, the catheter is easily passed in. Ultimately the tip of the appendix is cut off altogether, level with the skin if there is no wish to keep the appendicostomy open.

But if the appendix is to be used for injection for weeks, months, or even years, then a neat job can be made in the following way: Cut away the sero-muscular coat, as shown in the diagram (fig. 3), right down to the level of the skin, beginning with a circular cut at that level and continuing with a longitudinal cut. The sero-muscular coat then easily strips off. Then turn back the muco-submucous layers like a coat sleeve, and a neat little nipple results. This usually diminishes in size, the mucosa becomes skin-like, and the lumen is easily found when wanted at or near the summit of the nipple. When the meso-appendix is thick

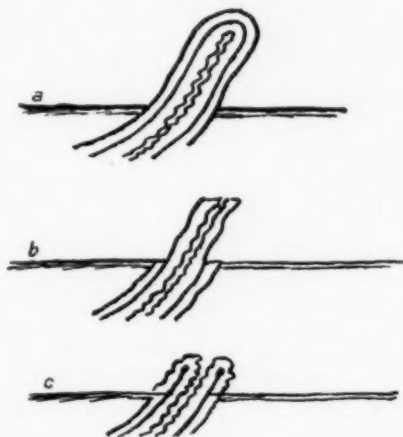


FIG. 3.

the aperture is sometimes lateral. In exceptional cases the mucous membrane prolapses a little. If so, it is easily trimmed off.

In order to reopen a transplanted appendix which has been disused for some time the procedure depends on the condition. If even a pin-hole opening remains it can be dilated, sometimes almost painlessly, even without local anæsthesia, first using a very fine probe, then a pair of sinus forceps, then a pointed or appendicostomy catheter. In such a case I have found the contraction nearly, if not quite, always confined to the skin, the middle and proximal part of the appendix remaining quite open.

Force should only be used in dilating the skin aperture by opening the blades of the sinus forceps. When once the soft rubber catheter is in the right place it may be pushed on boldly. But care should be taken to get it in the right place. The following awkward but, as it proved, harmless accident occurred: The appendix having been placed in the abdominal wall ready for opening, Dr. A. assisting and Dr. B. giving the anæsthetic, the place to open it was explained to Dr. A. But circumstances caused Dr. B. to take on the after-treatment of the case and he was unable to get the catheter into the cæcum. The operator found that the projecting end of the appendix had been notched on the wrong side and the catheter thrust into the meso-appendix. However, the antiseptic precautions had been good and so no harm was done.

It is a misfortune when necessity, or some other cause, hands the after-treatment of an operation case over to the anæsthetist instead of to the assistant, who has, of course, had the opportunity of seeing exactly what has been done, and generally also hearing and learning the wishes of the operator at first hand.

APPENDICOSTOMY FOR CONSTIPATION.

Appendicostomy is, I believe, the best surgical treatment for constipation that has yet been devised. So far it has proved with me unailing. One or two of the most obstinate cases of constipation I have met with or read of have yielded to it at once. And in the cases in which I have not done the appendicostomy for constipation but in which the latter trouble has existed or occasionally complicated the course of the case, the injection of diluted aperients, especially saline sulphates and carbonates or cascara, has speedily given relief. Often the mere injection of warm water through the appendix has sufficed.

An interesting and very important question is: *Will the subject of an appendicostomy for constipation have to use his appendix for the rest of his life in order to maintain regular action of the bowels?* I must write with reserve on this point because it is not yet even four years since the first appendicostomy was performed for constipation.

It has not been necessary to increase the strength of the purgative solutions through the appendix. On the contrary, it has generally been possible to diminish it. And sometimes it has been found that plain warm water in sufficient quantities ultimately sufficed. But progress in this respect has been slow.

I believe that a subject of constipation, if intelligent and willing to obey rules as to diet and exercise, even if it were found necessary

or advisable to do an appendicostomy, would, after that operation, have a better chance of being completely cured by an experienced physician than before. Strictly speaking, appendicostomy is not in itself a remedy for constipation, but an operation which facilitates the employment of remedies. One thing which an appendicostomy cannot help doing is to bring home to the subject the enormous value of taking enough plain water into the alimentary canal, and taking it at proper times. It can be further used to teach the lesson of the advantage of not drinking copiously at meals. Many people dilute the stomach, indirectly irritate the pylorus, and ultimately bring about constipation through taking much beer or whisky and soda at meals. They imagine that the food protects them from injury by alcohol. Really, their copious draughts at lunch and dinner aggravate the ill effects of their ill-chosen and excessive food.

The present condition of E. B., the patient on whom the first appendicostomy for constipation was performed three and a quarter years ago, is as follows: She secures a movement of the bowels when she chooses to take a suitable pill, or can find time to give herself a simple enema of soap and water *per anum*. This may not seem much of a triumph, but it should be remembered that before the operation the physicians could not obtain a movement of the bowels for long periods, in spite of the combination of rectal enemata with powerful purgatives. Her appearance and well-being are vastly improved. She is a factory girl. Were she of independent means I believe that, with bicycling or horse exercise and regulated diet, she would rarely need either purgatives or enemata.

Some of the slighter cases of constipation, in which the trouble was complicated with appendicitis or other disease, are now completely free from the need of enemata or the regular use of purgatives; but, of course, that improvement may be wholly or in part due to the cure of the coexisting conditions.

To the value of appendicostomy in the treatment of various forms of colitis ample testimony has been borne by various authors, and it is confirmed by cases in my own series. See, for example:—

In Case 8, of ulcerative colitis, appendicostomy transformed the condition of the patient from one of extreme emaciation and "typhoidal" apathy, in about three weeks, into a bright, vigorous, and beaming young man, with fat cheeks as round as apples, besides quite curing the colitis. Other cases were equally successful, though less rapidly so.

Doubt has been thrown on its value in mucous colitis. I have found

it of great benefit in this trouble. But, of course, particular regard should be had to the character and to all the symptoms of a person suffering from mucous colitis, which sometimes appears to originate in a defective state of the nervous system, and even to be itself a neurosis. Certainly if the patient has been a hypochondriac before the symptoms of mucous colitis appeared an appendicostomy cannot be expected to cure his hypochondria, unless the latter arose from auto-intoxication through the large intestine, with or without chronic constipation.

In intussusception the value of appendicostomy is obvious. It anchors the cæcum to the parietes, preventing recurrence; it permits immediate hot lavage of intestine, which is inflamed and bruised, and its peritoneal coat often torn by the intussusception or by its operative reduction; it checks hæmorrhage, washes away clots, decomposing fæces, and mucus. Its value as a means of recovering from shock may be disputed on the ground that hot water or neutral saline or peptonized milk can be as easily administered *per rectum*; but they are not in danger of being quite so quickly and completely returned when passed in through the appendix.

As a safety valve in association with excision of the ileo-cæcal valve it could, if necessary, have been utilized in Case 9. Of its use in typhoid fever there is as yet, so far as I know, no experience recorded. For the relief of intestinal distension in toxic conditions, apart from its use as a substitute for cæcal colotomy in intestinal obstruction, I have not found it of much value. But, more by ill luck than necessity, I have found a difficulty in getting nurses to use it correctly for the purpose. It is perhaps a mistake to try to combine the attempt with the occasional use of the appendicostomy as a means of passing fluids in. When this double trial is made, the tube or catheter is apt to be clamped, and *left clamped*, too long after each administration of an appendical enema.

On appendicostomy for the administration of nutrient enemata I have already commented.

APPENDICOTOMY AS CONTRASTED WITH APPENDICECTOMY IN THE TREATMENT OF APPENDICITIS AND OF CONCRETIONS IN THE APPENDIX.

By appendicotomy I mean incision of the appendix for the purpose of draining that organ itself, or of removing from it a foreign body or a faecal concretion. The first recorded "interim" operation for appendicitis, that of Mr. Charters Symonds, was an appendicotomy.

A concretion was removed, and the appendix, which was buried in adhesions, was left in situ in the peritoneal cavity. Symonds' operation was never repeated, so far as I know. It was ousted almost at once by appendicectomy. My opinion, as I have stated elsewhere (Cavendish Lecture, op. cit.), was that to put the operation of appendicotomy on a scientific basis it was necessary to combine it with transplantation into the abdominal wall, which, of course, removes it from the peritoneal cavity. This is precisely what was done in Case 16 of this series, and with entire success.

Appendicotomy may be either lateral, as in the case just quoted, and the incision closed by a suture after the removal of the concretion, as was also done in the same case, or it may be terminal. In the latter case it is, I think, better not to put in a suture, but to leave the opening to drain the transplanted and preserved but not quite healthy appendix. In Case 16, although I closed the lateral incision, I opened the appendix near its tip for the purpose of drainage and of dilating any stricture that might possibly exist. The decision to make a lateral opening or not should, I think, depend on the size, hardness, and position of the concretion, and on the state of the appendix itself. A small concretion, unless prevented by a stricture of the appendix, can be pushed towards either end of the appendix, and soft concretions can easily be kneaded and, as it were, milked along. I have performed both these manœuvres. But many concretions are large and lie in a kind of nest, sometimes with ulcerated walls. They are best cut out. They will come through a longitudinal incision smaller than their own diameter. A single Lembert's suture completely closed an incision through which was gently squeezed out a concretion nearly 1 in. long.

Malignant Disease of Stomach and Colon. Intestinal Obstruction.

Fæces passed through Dilated Appendix for Three Months.

First recorded case of the kind.

(1) Mrs. G. C., aged about 35; December 21, 1905. Intestinal obstruction due to malignant disease of the stomach involving the colon and peritoneum. Laparotomy; appendicostomy. Although the appendix was very slender it was stretched without difficulty to the size of a small rectal tube, and for three months the fæces passed through it, the intestinal obstruction being completely relieved. Warm water enemata *per appendicem* were given regularly. Ultimately, some of

the faeces passed *per rectum*. This patient was partly fed through a jejunostomy, and it was noteworthy that though some cutaneous eczema complicated that opening, there was none around the appendicostomy. Was this due to the alkaline secretion of the appendix bathing the outside of the rectal tube in the appendix? In March, 150 oz. of ascitic fluid were drawn off. Malignant nodules had been seen in the peritoneum when the appendicostomy was performed. Three and a half months after the appendicostomy the patient died. In the meantime she had been singularly free from physical pain and distress.

Advanced Ulcerated Malignant Disease of Breast and of Axillary Glands. Trunk covered with Large Canceroderms. Chronic Intestinal Obstruction and Melancholia. Appendicostomy.

(2) A maiden lady, middle-aged; with Dr. E. G. Younger; clever, reserved, and profoundly melancholic. Intestinal obstruction, apparently located in the sigmoid flexure, alternating with muco-membranous diarrhoea. On looking at abdomen we saw, forming a striking and tree-like pattern, numbers of large, brown, slightly raised stains, dotted here and there with bright red points. While discussing the case and deciding that these "canceroderms," as they have been called, confirmed the diagnosis of malignant disease of the colon, the nurse came in reporting a tumour of the breast which she had just discovered. This was a much-ulcerated but movable scirrhous of the left mamma, which the patient, though so intelligent, had concealed. The breast was removed and the axilla cleared out and the wound healed. An appendicostomy was done as a palliative measure after a laparotomy had discovered no intestinal tumour. The patient was difficult to feed, her melancholia got worse, and she died quite suddenly soon after leaving the nursing home. It was apparently the mental and moral effect of discovering that her complaint was cancer which struck down this sensitive and highly cultured authoress—a blow from which she never recovered.

Appendicostomy for Intussusception. First recorded case.

(3) J. T. H., aged 1 year and 10 months; April, 1905; Dr. Burstal. Ileo-colic intussusception, appendicostomy.¹

(4) Ellen B., aged 15; March, 1905; Dr. E. Burstal.² This was the

¹ Reported more fully in *Brit. Med. Journ.*, 1905, ii, p. 863.

² A name following the date is that of the house surgeon.

first case operated on for constipation. Her bowels would defy purgatives and rectal enemata for three weeks at a time.¹

After-history of this case brought up to date. The appendicostomy was used almost continuously for two years and nine months. For the last seven months it has been allowed to remain closed. The purgatives used *per appendicem* were mag. sulph., white mixture, and cascara, each largely diluted with hot water. The most efficacious she found to be ext. cascara. sagrada. liq. 2 dr., glycerini 3 oz.; $\frac{1}{2}$ oz. in not less than a pint of hot water at a time. She works as a tea packer from 8 a.m. to 8 p.m., and has been content with securing two or three actions of the bowels a week. In the last seven months, since she let the appendicostomy close, she obtains an action by injecting soap and water into the rectum. Pills, &c., by the mouth were, until recently, useless in this case. Both mother and daughter agree that injections *per appendicem* "answered much better than" rectal enemata. Her condition now, and for a long time, is quite different than that before the operation. She looks brighter, feels better, and has her bowels under control. If she had had time to use the appendicostomy daily I think she would probably have been quite independent of enemata.

She let the appendicostomy contract from disuse several times in the course of the first two years, but, after a fortnight or three weeks, always came to me to dilate it again for her. This was easily done with a pair of sinus forceps, as the contraction was only at the external end of the appendix.

Complete Intestinal Obstruction (without Stricture or Strangulation) in a Lady, aged 78. Appendicostomy. Appendix dilated enough to give egress to fæces at first, but now used simply for daily lavage of large intestine. Excellent health. An attack of perforative appendicitis due to fish-bones while the appendix is in the abdominal wall; trivial symptoms; quick recovery.

(5) Miss R., aged 78; with Dr. J. D. Roberts, of Ealing. This case was referred to in the Cavendish Lecture for 1907. Complete intestinal obstruction of some days duration; no flatus passed, no history suggestive of stricture; constipation. Laparotomy; appendicostomy. Appendix dilated to permit egress of gas and liquid fæces; great relief; copious hot water enemata *per appendicem* and *per rectum*; comfortable convalescence; daily warm injections through appendix. Excellent health

¹ Fully reported in *Brit. Med. Journ.*, 1905, ii, p. 863, and more briefly in the *Brit. Med. Journ.*, 1905, ii, p. 1358.

until, in June, 1908, an attack of pain in appendix with fever and constipation. Two fish-bones, each $\frac{7}{8}$ in. long, had perforated the appendix and caused a small abscess over it in the abdominal wall; on letting out the pus they came away; attack lasted only two or three days; recovery at once. It would be difficult to find any person of her age who looks brighter, healthier, or younger.

Subacute Intestinal Obstruction (Atony or Fæcal Impaction?) Appendicostomy. Perforation of Appendix while Buried in Abdominal Wall. Superficial Abscess. Recovery.

(6) Mary Ann R., aged 66; September 17, 1906; Dr. J. F. H. Dally and Dr. J. H. D. Acland. Subacute intestinal obstruction, cause not determined. Laparotomy; omental adhesions on left side. Appendicostomy; patient very fat. After a few days, suppuration in the fat over the appendix, which was found to be perforated. Was this perforation pathological or traumatic (*i.e.*, by a probe)? Wound opened superficially and appendix slit up from apex to perforation; recovery. Appendicostomy used to wash out large intestine periodically.

Mucous Colitis, Severe and of Long Standing. Appendicostomy. Cure.

(7) G. L. S., aged 32; March 18, 1907; a patient of Dr. Broadbent, of Collingham; a farmer, broad-shouldered and strongly built; very anæmic-looking; worn out with chronic diarrhœa and "indigestion." Mucous colitis. At Dr. Arthur E. Saunders's suggestion, appendicostomy was performed. At the laparotomy the cæcum, transverse colon, and sigmoid were all found markedly hypertrophied, as if thickened by chronic inflammation. A large catheter was placed in the proximal part of the appendix and the distal part cut off. Sloughing took place nearly up to the cæcum, which converted the operation almost into a cæcotomy. For a long time, therefore, the patient had a difficulty in keeping himself perfectly clean, but he succeeded ultimately, as the wound healed and contracted. He still, after one year and three months, uses the appendicostomy. The injections used were of protargol solution, alternating with hot water and weak solution of borax and sodæ bicarb. with common salt. Progress was rapid. In April, after returning home, he wrote: "I can tell my bowels are gradually improving, scarcely ever being moved more than twice in the twenty-four hours, and that after the injections"; "I am still using the second mixture you gave me, not the protargol." Now in good health, but occasionally gives himself hot water injections at bedtime. No symptoms of colitis.

Acute Hæmorrhagic Colitis: Twelve to Fourteen Stools a Day with much Blood in Them. "Typhoidal" State. Appendicostomy. Rapid Cure.

(8) Arthur S., aged 23; October 6, 1907; Dr. C. Tyler. Colitis with hæmorrhage, ulcerative (?). Had suffered from pain in "stomach" and bleeding from rectum for seven days; fifteen stools a day, often bloody; vomited on admission. Looks very ill, pale, with eyes sunken, tongue furred; temperature 99.6° F., pulse 98; very apathetic and difficult to get a clear history from. Rigidity, tenderness, and limited movement on right side of abdomen. No Widal's reaction. October 7 to 24: Treated by enemata of protargol, 1 in 4,000; bismuth, opium, tannalbin; starch and opium enemata; milk, arrowroot, Benger's food. Improvement at first, but not maintained. Transferred by Dr. Beddard to surgeon. Sigmoidoscope showed red, granular, bleeding state of sigmoid mucosa. Stools now bloody, eleven to fifteen a day; patient very emaciated and apathetic. October 29: Laparotomy; large intestine, including cæcum, thickened, congested, and covered in places with marks resembling patches of lymph in appearance, but *in* the serosa not *on* it. Appendix in a similar state; 6 in. long and everywhere closely bound down to cæcum; appendicostomy; immediate catheterization; sodæ boratis, sodii chloridi, of each $\frac{1}{2}$ dr., water 20 oz. With this (hot) large intestine washed through twice a day; 30 oz. of protargol solution ($\frac{1}{2}$ gr. to 1 oz.) once a day, soon increased to 1 gr. to 1 oz. twice a day. Rapid improvement. Quite well; discharged within a month very fat and strong, and remained so.

Excision of Ileo-Cæcal Tuberculosis. Appendix arranged for use as a safety valve, if needed.

(9) D. F., a girl, aged 14; May 10 and May 12, 1907; Dr. Gilbert Richardson's case. Tuberculous disease of ileo-cæcal valve and of ileum immediately above it. Excision at two stages. July 3: The ileum was sutured to the cæcum, and the appendix left in the wound for use as a safety valve, if necessary. The patient did well in every way and is the picture of health. My recollection is that this use of the appendix proved quite superfluous in this particular case, but it might have been of use, and it did no harm.

Recurrent Appendicitis, Acute. Appendix Transplantation. Recovery.

(10) Thomas P., aged 46; September 20, 1906; Dr. Dally. Recurrent appendicitis; third attack this year, laid up six weeks with first; abdomen not moving well, tenderness, severe pain, rigidity; symptoms subsided with treatment. October 2: Appendix transplanted obliquely into abdominal wall, tip being drawn through a special opening in skin, &c., above prime incision; appendix very long and vascular; pain the first night; a little subcutaneous pus had to be let out of wound on the thirteenth day. After operation, temperature 98·6° F., pulse 80, respiration 24 to 20; they were the same before operation. Temperature never rose above 99·2° F.

Pelvic Troubles and Operations. Muco-Membranous Sigmoiditis and Gall-stones. Much pain. Morphia habit. Cholecystotomy and Appendicostomy. Greater part of Appendix Transplanted. Afterwards Cholecystogastrostomy. Cure.

(11) Female, aged 57; with Dr. A. L. Curtis, Dr. T. W. Bailey, and Dr. J. M. Bennion, of Orpington and St. Mary Cray. This lady had been an invalid for seven years, suffering mainly from severe paroxysms of abdominal pain, which appeared to her to be due to the passage of wind or fæces through a coil of bowel in the region of the sigmoid. This spot was tender; long history of muco-membranous colitis; left ovary had been removed; patient worse afterwards; sent by a most eminent surgeon to Plombières. A medical man there, instead of giving the Plombières treatment a fair trial, sent her to a surgeon in Paris for operation. In Paris, told that operation, apparently of the nature of intestinal resection or short-circuiting, was essential; that it should be done at once; and that to travel to London before it was done might be fatal. She took the risk (viz., that of travelling home). Afterwards had severe attacks of jaundice. I first saw her in one of these. A tall, well-built patient, but with an enormous fat-laden abdomen, very relaxed, jaundiced, and compelled to take morphia (4 gr. a day) for pain, much mucus and membrane in stools.

Two operations.—(1) For stone in common duct. Small, round movable calculus felt distinctly, but, having let it go in order to show assistant how I wished the very fat viscera to be held out of the way, I could not find it again. Drainage of gall-bladder; no tumour or stricture of sigmoid felt. At the same time, appendicostomy; regular injections

through the appendix of argyrol solution of hot water. Patient did exceedingly well, but as biliary fistula remained and bile in due quantity only appeared intermittently in the fæces, (2) a cholecystogastrostomy was performed. The duodenum would not come to the gall-bladder without tension and trouble, and the stomach lay invitingly in apposition; Murphy's button. Result in all ways excellent; patient restored now for more than a year to health, to society, to family life, and free from morphia habit.

Remarks.—I think that, at the time it was recommended, the Plombières method of treatment might have cured the patient, and it is remarkable that after she had been sent by an English surgeon to that place, a Plombières practitioner should have rejected the diagnosis and sent her away to a surgeon in Paris.

History of many attacks diagnosed as Appendicitis. Laparotomy. Transplantation of Appendix. Cure.

(12) Elizabeth W., aged 15; December 6, 1907. This girl was sent over from the medical side with a history of many attacks of "appendicitis," marked by vomiting, pain, inability to pass urine, and the appearance of a swelling. As each attack had only lasted half an hour, as there were at present no physical signs, except a small lump above Poupart's ligament, and as her little sister who had been a patient in hospital with incipient hip disease had greatly enjoyed her stay, I doubted the genuineness of this case, and for some time hesitated to operate. However, her complaints were repeated, so she was admitted and, lest I might overlook some incipient tuberculous or other affection, on December 10 I operated on her. The lump was found to be a slightly enlarged gland. Incision over appendix. Pelvis explored and its contents found normal; cæcum and appendix apparently healthy, the latter rather long. The appendix was transplanted into the abdominal wall, so as to lie obliquely with its apex upwards, brought through a special button-hole in the skin. Local pain for twenty-four hours; headache for three days. Ten days afterwards, although the appendical artery had been divided, the tip of the appendix was projecting 1 in. looking healthy and rosy. It was afterwards cut off. No pain.

Like most of my appendix operation cases she had, for some time, hot water enemata every four hours (in this case 6 oz.). This operation caused rises of $\frac{1}{2}$ ° F. in temperature, of 8 beats in the pulse, and of 4 in the respiration. The patient occasionally calls at the hospital, apparently

quite well, but sometimes with slight complaints not located in the appendix.

Acute Inflammation of Appendix Vermiformis and of Right Uterine Appendages. Transplantation of Appendix. Right Ovary and Tube at same time moved from Douglas's Pouch to the Brim of Pelvis. Cure.

(13) Florence M., aged 22; December 12, 1907; Mr. Tyler. Single. For five days pain in the abdomen, worse on right side. Began with vomiting. Abdomen tender, rigid, not much distended; no dulness.

Operation.—Battle's incision, but lower than usual, to permit pelvis to be examined. Appendix vermiformis freshly inflamed; a reddish rough area of 2 sq. in. on cæcum; tip of appendix coiled sharply on itself; right ovary and tube prolapsed into Douglas's pouch, very thick and dark red; between the two much adherent dirty white lymph. The appendix was transplanted, with its apex carried through a button-hole in the skin above the parietal incision; the right uterine appendages were brought over the brim of the pelvis, where, being very stiff and thick, they remained in contact with the lower end of the parietal wound. Two tubes and a gauze drain to bottom of pelvis. Another gauze drain in the position of the adherent dirty white lymph. Some vomiting and pain in first twenty-four hours; then rapid convalescence. On admission: Temperature 100.4° F., pulse 84, respiration 24. After operation, temperature 100° F., pulse 92, respiration 24.

Many Attacks of Appendicitis. Abscess in Scarpa's Triangle. Appendix Transplantation. Cure.

(14) Florence D., aged 31; January 21, 1908; Dr. Tyler. Repeated attacks of appendicitis between nine and six years ago; then a free interval of five years; then recurrence with abscess formation. Eleven days ago pain in pelvis and thighs. January 18: A rigor, and in right groin a swelling below Poupart's ligament. January 27: This was opened by the house surgeon; pus fæulent.

Operation, January 28.—Battle's incision; extensive adhesions; cæcum flattened down and fixed in iliac fossa; appendix stretched away beneath it downwards to external iliac artery and Poupart's ligament, to both of which it was firmly adherent. Its bulbous and perforated apex led into the sinus left beneath Poupart's ligament by opening the abscess in the groin. The appendix was freed and transplanted

with the apex upwards into the upper end of the parietal wound. The fossa from which the cæcum and appendix had been dislodged by operation was temporarily packed with gauze. The end of the appendix when cut off showed an impervious stricture, 1 in. from the apex. A pint of warm water was injected through the appendix daily. Pain for about forty-eight hours. On thirty-fourth day after operation discharged; very well. Temperature after operation 100° F. once only, then normal; pulse sank steadily from 112 to 72 in ten days; respiration from 26 to 20.

Acute Appendix Abscess. Pelvic Abscess. Operation. Appendix Transplanted; opened at once, and catheter left in. Appendix sloughed and was excised. Recovery.

(15) Beatrice G., aged 15; March 6, 1908; Mr. Tyler. Appendicitis with mass in iliac fossa and behind pubes; first attack, three weeks' duration. Temperature 102.2° F., pulse 136, respiration 40. Abdomen moved badly, extremely tender, much pain; patient looks very ill.

Operation.—Battle's incision, extending well downwards; mass of thick adherent omentum clamped and divided; omentum, right uterine appendages, and a large mass extending deep into pelvis. General peritoneal cavity packed off with gauze; much foul pus evacuated from swelling with patient turned on her side; careful swabbing. Appendix passed deeply into pelvis; it and cæcum firmly adherent to surroundings; they were carefully separated with swabs and the finger, but the extreme tip of the appendix torn off; much oozing. The cæcum and appendix were brought up, and the latter placed in the upper part of the wound in the abdominal wall, with its base fixed by a couple of catgut sutures. Packs which had been used to stop bleeding were now removed, and a large gauze drain carried right down into pelvis to site from which appendix had been removed. The middle of the wound was closed with stout catgut sutures enough to prevent escape of intestine, but not elaborately. The appendix was opened at once, a catheter tied in, and a pint of neutral saline injected. Both pulse and respiration were better after than before operation, and the next morning had dropped to 108 and 32 respectively, and continued to descend to 80 or 90 and to 20 or 24.

General condition: Second day much easier; third day improving steadily, but the appendix was sloughing.

Second operation.—March 15 (fifth day): Wound reopened; appendix

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cut off and hole in cæcum closed with sutures, two rows; middle of wound in abdominal wall closed over this; ends left open.

A small fæcal fistula was left, and on April 7, without opening the general peritoneal cavity, this was successfully closed by operation. However, on May 11 a small abscess formed and broke in the scar, and raised her temperature to 100·2° F. This broke and healed in a fortnight. She has been seen repeatedly since then in good health.

Recurrent Appendicitis. Concretion in Kinked Appendix removed by Appendicostomy. Appendix, except Kinked End, Transplanted. Cure.

(16) Edward G., aged 16; December 30, 1907; Dr. Tyler. Had had four attacks, diarrhœa in each. In bed eight weeks with one.

Operation.—Battle's incision; a few recent adhesions; appendix was kinked at the tip and contained a concretion. A slit was made in the side of the appendix and the concretion removed. The slit was closed with a single suture of fine catgut. The appendix was then transplanted into the abdominal wall obliquely, and the kinked end cut off in such a way as to leave the mucous coat longer than the sero-muscular (fig. 2). Then the mucous coat was doubled back like a coat cuff till its cut edge touched the edge of the skin wound; the sero-muscular coat had been divided exactly level with the skin. The result was a very neat little mucous papilla, of course open in the centre.

HIGHEST RECORDS.

Before operation			After operation		
Temperature	98·2° F.	...	99·6° F.	once, then normal.	
Pulse	...	76	...	112, normal from third day.	
Respiration	20	...	20,	always normal.	

In hospital thirty days, always well, and remained so after discharge.

Gastritis and Appendicitis (Traumatic?). Appendix Transplanted. Much Benefited.

(17) Thomas S., aged 28; December 12, 1907; Dr. Tyler. Had long complained of vomiting, on an average two hours after food, and of pain and tenderness in the region of the stomach, liver, and ascending colon. Had ague in India, but attributed the present illness to a blow on the right lower ribs with a steel spanner 2 ft. long. Abdomen somewhat retracted in upper part. Patient looks ill; temperature 99° F., pulse 64; tongue furred. Laparotomy through upper part of right rectus sheath;

peritoneal coat of stomach congested; other things normal. Second incision over appendix; it was congested like the stomach; transplanted. Patient quickly and steadily improved. In March he was better, and in May (six months after operation) much better and at light work. Diagnosis: Reflex trouble due to chronic catarrh of appendix following injury (?).

Recurrent Appendicitis. Short, Kinked Appendix Straightened Out and Transplanted. Well for Seven Months. Then Pain in Region of Gall-bladder. Colitis (?). October 6 (eleven months after operation): Appendix opened and douching commenced through it. Symptoms removed up to date.

(18) Elizabeth B., aged 33; married, no children; November 9, 1907; Dr. Tyler. Attacks of pain in right iliac region for eight years, in bed five weeks with one; pain worse at catamenia; for three weeks had vomited all solid food; diarrhoea; tenderness. Laparotomy; right ovary normal, but appendix, 3 in. long, kinked and beneath cæcum. It was straightened out and transplanted with tip projecting beyond skin. The projecting appendix tip dried up and separated, and the lumen was allowed to close. June, 1908 (seven months after): Has been quite free from pelvic and right iliac pains ever since operation, but has lately had a feeling of discomfort and tenderness over the gall-bladder and is inclined to be constipated. October 6, 1908: The discomfort, &c., in the gall-bladder continuing, but there being no other signs of gall-stones, the appendix was opened and douching through it commenced. November 5: Has been out and about three weeks and feels quite well.

Recurrent Appendicitis, Frequent Attacks. Appendix Transplanted. Cure.

(19) Harold W., aged 13; June 12, 1907; Mr. F. J. Treves. Recurrent appendicitis, frequent attacks; pain always localized in right groin; tenderness. June 14: Appendix transplanted into abdominal wall. The diagram in the text is from this case. Fifth day: Appendix tip cut off. Twenty-seventh day: Left hospital well. "End of appendix appears as a small red projection with no discharge from it." Bowels regular. After operation highest temperature 99.2° F., pulse 112, respiration 28.

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Intussusception Reduced by Laparotomy. Appendix Transplanted. Recovery.

(20) William B., aged 4; 1907; Dr. Tyler. Ileo-colic intussusception, reduced by operation; appendix transplanted into abdominal wall; tip cut off. October 22: Prolapsed mucous membrane snipped off. Pad and bandage. Quite well.

Severe Appendicitis. Suppurative Peritonitis, Drained in Loin and per Rectum. Enterostomy. On the eighteenth day the enterostomy closed and the proximal $3\frac{1}{2}$ in. of the Appendix Transplanted. Cure.

(21) William E. W., aged $8\frac{1}{2}$; March 15, 1907; Dr. Acland. After a very severe attack of suppurative appendicitis in which much pus had been evacuated from the appendix region, from the right loin, and from the pelvis, followed by rectal drainage and an enterostomy in the iliac incision, to relieve distension as well as to feed, on the eighteenth day the enterostomy opening was closed by suture and, at the same time, the proximal half of an appendix which was 7 in. long and kinked distally was transplanted obliquely into the abdominal wall. Final result perfect and remains so; seen October 8, 1908 (nineteen months after).

Gangrene of Upper Coils of Jejunum. Operation: Appendicostomy for Nutrition. Death.

(22) George D., aged 41; April 19, 1908; Mr. F. P. Young. Had drunk "2 or 3 pints" of beer daily. He was found at the operation to be rather fat, but to have an exceedingly large abdominal cavity. For three months there had been, twice a week, acute attacks of paroxysmal pain above the pubes, causing vomiting and sweating. No hæmaturia. Micturition normal. Last attack worse; began on April 19; bowels open same afternoon. Patient pale, covered with sweat. Temperature 98° F., pulse 80, respiration 26; next day temperature 103° F., pulse 116, respiration 30. April 21: Transferred to surgical side and *operated on*. Median incision; foul pus welled out; pelvis was swabbed clean; small intestine much inflamed, slightly distended, and nowhere empty. Appendix normal. An enterectomy was performed with Paul's tube and an appendicostomy also done (for nutritional purposes). Patient died next day. It was found post mortem that the prime trouble had been overlooked. Owing to the patient's condition, the operation had to

be hurriedly finished. There was gangrene of the small intestine, high up in the jejunum and above a stricture.

The time occupied in doing appendicostomy here would have been better spent in exploring more carefully the abdomen above and to the left of the umbilicus, where the gangrenous intestine was located. The general distension of the small intestine caused me not to suspect a stricture high up in the jejunum, but the fact that the vomiting had not been faecal might have suggested the truth.

Acute Ascites in an Infant aged 6 weeks. Great Emaciation. Food Refused. Appendicostomy for Nutrition. Death.

(23) Harold H., aged 6 weeks; November 29, 1906; Dr. Acland. Acute ascites, cause unknown, great emaciation; food refused; temperature on admission 97° F., pulse 144. Laparotomy; much pale yellow fluid evacuated; no tubercle seen. Appendicostomy for injection of neutral saline, &c. Death on second day. No post-mortem.

Perforation of Appendix. Extensive Suppuration. Drainage per Vaginam. Appendicostomy for Nutritional Purposes. Death after Secondary Operation.

(24) Annie K., aged 27; December 19, 1906; Dr. Acland. Extensive suppurative peritonitis; perforation of appendix. Illness began acutely seven days before admission; complete obstruction, no flatus for two days; vomited everything. Seven years history of indigestion and constipation. Median laparotomy; foul pus "poured" out; dry swabbing; drainage through vagina as well as anteriorly. Second incision above umbilicus; no pus in perigastric region. Anaesthetic, gas and oxygen only. On eighth day a urinary fistula formed. "Patient has a cough, but is getting better." On twenty-second day faecal discharge through abdominal wound. Less urine through fistula, more *per urethram*. Some expectoration. On thirty-sixth day appendix freed from dense adhesions; much pus let out and appendicostomy done at end of operation because of low condition of patient. One pint of hot neutral saline through it every four hours. Death on third day from exhaustion.

Radical operations are dangerous in such cases. The temptation to them is the desire of shortening the case and the fear of exhaustion and of secondary infection; but it is, perhaps, better to trust to careful drainage and patience.

Acute Perforative Peritonitis. Faecal Pus in Peritoneal Cavity. Bad General Condition. Appendicostomy for Nutritional Purposes. Enterotomy for Obstruction. Death.

(25) Lawrence F., aged 11; May 29, 1908; Mr. Young. A severe case of acute purulent peritonitis with faecal-smelling pus in the general peritoneal cavity, probably arising from perforation of the appendix, which was found lying quite free with no adhesions. Operation day of admission; pelvis opened through median incision; appendix brought out through an iliac opening. Gauze and tube drainage through both incisions. Appendicostomy for nutritive purposes and neutral saline injections given through it, as well as peptonized milk. Vomiting and delirium continued from time of operation till death, about forty hours afterwards.

An enterotomy was done by simply incising a coil of intestine lying beneath the iliac wound. Green fluid similar to the vomit escaped; but this enterotomy was too late, as the patient was already sinking.

Old Chronic Pelvic Suppuration. Appendix and Cæcum in Douglas's Pouch. Operation: Rectal Drainage. Death. The Appendix arranged for Appendicostomy but not used.

(26) A male patient, aged about 35, who had for years been an invalid with some chronic abdominal affection, accompanied by nightly rise of temperature. Great wasting. A median laparotomy above the pubes discovered not only the appendix but the cæcum fixed firmly in the bottom of Douglas's pouch, and between them and the rectum and bladder pus and cheesy matter. The adhesions were separated, the cæcum brought up to the laparotomy wound and the appendix into the wound, the abscess cavity drained both by a tube into the rectum and by a tube and gauze from the original wound. The Trendelenberg position was used. For two days the patient seemed to do well. On the third he rapidly changed and died.

Study of his chart and history make it probable that he died of exhaustion which might have been prevented by putting enough water into his vessels. I made a mistake in draining *per rectum*. The drain, brought out at the anus, was the cause of rectal enemata of water or neutral saline not being given; and the appendix was never used for the purpose. It had been left in the wound ready to open if necessary, but the patient seemed to be doing well without its employment. Being

200 miles away I had little or no part in the after-treatment, and the latter was not conducted by the surgeon who assisted me at the operation, and who heard my views, but by his partner who gave the anæsthetic. My experience is that this change of duties at so critical a time as the commencement of the after-treatment is a dangerous mistake. In the only other case in these series in which it occurred I pointed out to the medical man who had assisted at the operation the exact spot where the appendix was to be opened. But the opening was deputed to another medical man, who opened, not the appendix, but the meso-appendix, and, of course, failed to pass the catheter. In some cases, after the parts have granulated, the swollen meso-appendix is difficult to distinguish from the appendix. The simplest plan then is to slice the top off both. The mucosa-lined lumen will then be obvious.

I ought, in the case I have narrated, to have simply packed the pelvic site from which the cæcum had been lifted with iodoform gauze, bringing the gauze and a rubber drain out of the suprapubic opening. Then suitable position and frequent injections of hot water *per rectum* or *per appendicem* would have most likely tided the patient safely over the dangerous early days.

No Movement of the Bowels for Three Months. Appendicostomy and Injection through Appendix. Quick Relief.

(27) Mrs. M., a patient of Dr. Crombie's, of Sidcup, whose case has been reported in the *British Medical Journal* for October, 1905 (op. cit.). Intractable constipation; she had passed no motion for three months; occasionally she regurgitated fæces and vomited them. She was carefully watched by a good nurse and others. Appendicostomy. Injection through the appendix of saline purgatives and hot water; an immense stool passed, but with great difficulty, the patient and the nurse having to help with hand pressure on the abdomen. The constipation formed only a part of this patient's ailment. Trivial wounds in her ulcerated, sloughed, and were got to heal only with great difficulty. She ought to be isolated from her friends, but will not consent, although that plan has been tried once with her successfully. But about the good effect on her bowels there was no doubt.

Appendicitis, Recurrent. Chronic Constipation. Appendicostomy.

(28) Alice S., aged 32, married, no family; in the West London Hospital, June, 1906. Had had two bad attacks of appendicitis. Had

suffered from constipation "as long as she could remember." Appendicostomy. Appendix opened on fifth day. She still (November, 1908) keeps her bowel regular by passing an appendicostomy catheter and administering through it injections of hot water or of hot water with extract of cascara. If she omits to pass the catheter for a day or two she has a difficulty in getting it in. The general health has much improved. No pains.

Extensive Adhesions round Appendix Separated and Appendix Transplanted with a view to Appendicostomy, but found impermeable. Recovery.

(29) Harriet B., aged 31, single; in the West London Hospital, October 31, 1905. In last two months repeated attacks of pain in appendix region, very acute and with vomiting. Had been operated on nine months before to separate old adhesions of the anterior wall of the stomach to the liver and anterior abdominal wall, and had been feeling well since, till two months ago. Tenderness over appendix.

Operation.—Many adhesions of cæcum, neighbouring small intestines, and omentum found out and separated, enough to permit appendix to be brought out and fixed in the abdominal wall with a view to performing appendicostomy. But the lumen would not admit the smallest catheter. The appendix was left in the abdominal wall. Good recovery, except on second day; pulse, respiration, and temperature unaffected by operation. After-history not followed.

Inflammation of Appendix Vermiformis and of Right Uterine Appendages during Pregnancy. Premature Labour. Separation of Adhesions and Transplantation of Appendix. Immediate Relief and Cure.

(30) Annie H., aged 32; admitted to the West London Hospital, July 11, 1908, eight days after confinement; for last month pain from pubes up to the costal margin on right side, with vomiting; labour at the sixth month, difficult and eighteen hours long. Constipation lately; looks ill; tongue dry; skin sweating. Slightly distended. Visible peristaltic movements; no rigidity; soft gurgling mass felt in right iliac fossa, probably the cæcum; deep tenderness; some dulness in right flank. *Per vaginam, nil.* Temperature 99.6° F., pulse 96, respiration 28. Albumin and pus in urine. A small mass can be felt in right iliac fossa and a band can be felt running from anterior superior iliac spine towards middle line; the small mass can be pushed inwards towards pelvis.

Operation, July 24, 1908.—Battle's incision. Right uterine appendages found thickened and inflamed and adherent to cæcum and appendix; the adhesions were separated and the appendix transplanted into the abdominal wall. The next day the patient was quite comfortable, but had a little pain in the night. Appendix was then opened and irrigation through it with hot water several times a day was commenced. A four-hour chart showed no increase of pulse, temperature, or respiration after the operation. Uninterrupted recovery; left hospital on twenty-fourth day. Keeps well.

Acute Recurrent Appendicitis. Old Strong Adhesions. Appendix Transplanted from Pelvis. Cure.

(31) H. R. C., a railway signalman, aged 30; under Dr. Bailey in Cray Valley and Chislehurst Cottage Hospital. Had had a previous severe attack of appendicitis and been in hospital once before with it. On July 2, 1907, was taken to the hospital with an acute and "violently painful" attack, and operated on next day. Many old, firm, and extensive adhesions were separated, and the appendix, which stretched over into the pelvis, transplanted into the abdominal wall. Uneventful recovery. On October 22 this year he wrote giving an excellent report of himself.

Subacute Appendicitis. Appendix sharply Curved, Adherent, Twisted, Strictured, and containing soft Concretions. Transplantation. Appendix remained inflamed until opened near tip. Rapid Cure.

(32) Francis D., aged 17; in the West London Hospital, May 4, 1908, with subacute appendicitis. Temperature 99·6° F., pulse 110, respiration 28; pain, tenderness, vomiting; constipated by habit; thickening felt in iliac fossa. After a week, temperature 99° F., pulse 60, respiration 20.

Operation, May 12.—Battle's incision; appendix very red, stiff swollen, in fact much inflamed; contained soft concretions (?); adhesions between appendix and cæcum and a kind of sheath over the proximal part of the appendix (fig. 4). That organ was freed and transplanted. Temperature rose on third day to 101° F., with much pain. Appendix opened, letting out muco-pus. Next day and subsequently no pain. On the seventh day, temperature rose to 103·2° F., and a little pus

escaped from wound. On the eighth day with a rubber catheter a stricture was detected. Hot water injections through appendix. Temperature came down to normal same day, and three weeks after the operation patient went out well and has remained so.

*Muco-membranous Colitis. Constipation. Appendix Transplanted.
Great Improvement. Still under Treatment.*

(33) Mrs. H., aged 35; one child aged 14; with Dr. Frank C. Ford. Vaginal discharge before and since confinement. Habitual use of injections of zinc. Attack of severe pain while in a tram, October, 1907; could not get off the car for the pain; in bed for some weeks. Two attacks since, chiefly over right iliac and right sacro-iliac regions. No vomit. Constipation. Blood and mucus in stools occasionally. No fever (?). Quite disabled.

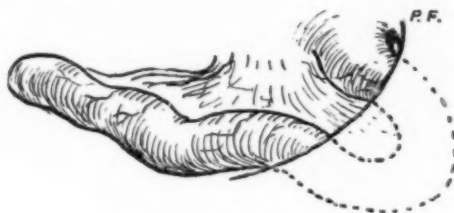


FIG. 4.

Appendix of Case 32 (Francis D.).

Operation, April 6, 1908.—Battle's incision (low); omentum extensively adherent in right lumbar region. Transverse colon hanging in a festoon in normal position of cæcum, held there by omental adhesions. Cæcum in pelvis full of fæces in round nodules; not contracted on them. Appendix healthy-looking, a little fæcal matter in it. Adhesions freed; appendix transplanted.

After-course satisfactory, but cure delayed by intolerance of silver salts. Appendical injections of borax, carbonate of soda, and salt solutions did good locally; but the patient's strength did not materially improve until she used plain hot water. Mother and daughter are just now laid up with scarlatina.

Enteritis of Lower End of Ileum : cause undetermined. Simulation of Appendicitis. Transplantation of proximal half of Appendix (6 in. to 7 in. long.) Appendical Injections. Cure.

(34) Mercy W., aged 26, a nurse; October 1, 1908; Dr. McLean. History of chronic constipation, not infrequent "bilious attacks," and irregular menstruation. For last month frequent headaches; seven days ago vomiting excessively, very ill, but in pain; vomiting continued until yesterday; "excessive" tenderness in right iliac region; pain yesterday. Operation day of admission; lower part of small intestine for 6 in. or 8 in. above ileo-cæcal valve red, inflamed, and thickened, with lumps both in its wall and in its mesentery (six or eight, each the size of a haricot bean, or larger, and not sharply defined); appendix normal, transplanted into abdominal wall. The temperature (102° F.) and pain gradually subsided, the former in a fortnight. Appendix opened on fourth day, but systematic injections not commenced until about fourteenth day; on seventeenth day a threatening of thrombosis in left leg, which, however, passed off harmlessly. With regular appendical enemata she rapidly improved and went home convalescent on the thirty-fourth day. Widal's reaction negative.

CÆCOSTOMIES.

In the period covered by these appendicostomies I have done two cæcostomies.

Chronic Colitis. Intestinal Obstruction. Appendicostomy. Great Improvement.

(35) Mr. H., aged 65, April 1, 1906; with Dr. Spofforth, of Cricklewood. Intestinal obstruction occurring in the course of a chronic colitis; complete relief; health afterwards greatly improved under a course of injections through cæcostomy. After some months the patient felt so well that he raised the question of having his cæcostomy closed, but it was decided to leave it open.

Cæcostomy performed for Constipation, because Appendix had been already Removed.

(36) A young woman named Florence F.; in the West London Hospital for obstinate chronic constipation. A cæcostomy done in the same manner in which Witzel does gastrostomy. Appendicostomy

would have been preferable, but the appendix had been removed a year before. This patient never learnt to manage her cæcostomy; could not even pass her own tube, although the cæcostomy would admit a lead pencil. Her medical attendant used to have to pass it for her. She allowed it to close up, and afterwards said she wanted to have it reopened as soon as she could get leave of absence from her duties. However, she is not nearly so constipated as she used to be.

DISCUSSION.

The PRESIDENT (Mr. Warrington Haward) said the Section would agree that the best thanks of its members were due to Mr. Keetley for such an interesting paper, and one which obviously furnished many points for discussion among both surgeons and physicians. Among them was the question of the value of appendicostomy in connexion with constipation and colitis, and the important question of the removal or transplantation of the appendix.

Mr. LEONARD BIDWELL said he had not been convinced that any kinked or perforated appendix was worth preserving. He thought the condition of a patient after removal of a kinked appendix would be preferable to that of one in whom it had been transplanted, since in the latter case a mucous discharging sinus was left. Most patients did not like this. After simple incision of an appendix abscess a sinus sometimes persisted, through which a little mucus was discharged, and in such cases patients usually wished to have something further done. He thought that after transplantation the patients might ask for the cure of the sinus. With regard to fixing the appendix in cases of intussusception in young children he was sure the success of an operation depended almost entirely upon the rapidity with which the operation could be performed. In the case of a child under 1 year the extra ten minutes required for fixing the appendix must influence the chances of recovery. He regarded appendicostomy as very valuable, and it was at present the operation of choice in intractable constipation. The cases he had seen in which that had been done had been most satisfactory, as were those recorded by Mr. Keetley. With regard to its use in mucous colitis, in more than one case in which he had intended to perform appendicostomy he had found evident disease in the appendix. On removing the appendix the symptoms of mucous colitis were cured. Thus some supposed cases of mucous colitis were due to chronic inflammation of the appendix. He was very glad Mr. Keetley had brought up the subject.

Mr. LOCKHART MUMMERY said the Section owed much to Mr. Keetley for having popularized the operation of appendicostomy, and for bringing forward his cases at a time when the operation was being extensively done. He thought the most important thing was to know how to get the best sort of opening in

such cases. One or two things might happen which could interfere with the usefulness of the operation. Mr. Keetley said he did not consider it absolutely necessary to preserve the meso-appendix or the appendicular artery. He (Mr. Mummery) had seen twelve or fourteen cases, and the only ones in which bad results had followed as regards the opening were those in which the surgeon had divided the meso-appendix. It was most important to avoid sloughing of the appendix. He saw recently a patient who had been operated upon for colitis by appendicostomy, the operation having been done in the country, and the operator had probably not had much experience of that operation. There was a faecal fistula into the cæcum, and she had to wear an apparatus to prevent the faeces from escaping at the opening. That should never follow a proper appendicostomy, and he wrote to ask the doctor how it was done. The doctor replied that he had divided the meso-appendix, and the entire appendix had sloughed out on the third day. He agreed with Mr. Keetley about not leaving the catheter in. Many cases had sloughed on that account, and he thought a catheter should never be left in. It was often asked how long irrigation should be continued where operation had been done for constipation. His own experience of that was confined to about six cases. One of them he operated upon one and a half years ago, and the patient no longer required the appendicostomy opening, and he thought it would be safe to close it up. He wished to be sure that no reopening would be required. He regretted to hear the expression "surgical treatment of constipation." Surely constipation was no more a disease than was vomiting. There were many kinds of constipation, and the surgeon never treated a symptom—he treated the cause. There were many cases of constipation in which appendicostomy was the ideal operation, but there were also many for which it was useless. The cause might be adhesions tying down the bowel, or a partial or chronic volvulus, and then appendicostomy would be useless for it. The same might be said about chronic mucous colitis. The cases in which the operation was useful were those in which there was a chronic inflammation of the mucous membrane of the colon. These cases could be diagnosed by the sigmoidoscope. In some cases the colitis was due to bad visceroptosis. Mucous colitis was no more a disease than was constipation. Before appendicostomy was performed, or at the same time, there should be an exploratory laparotomy, so as to ascertain the condition of the colon, if the diagnosis had not previously been made by means of the sigmoidoscope.

Mr. S. A. KIRKBY-GOMES said he thought surgeons working in the tropics owed very much to Mr. Keetley for his interesting paper, especially that part dealing with chronic constipation. He had had ten years' experience in the tropics, where one often had to treat European ladies, principally for chronic constipation. They were a class of people who were intelligent, but unwilling to obey the rules of diet and exercise. No doubt the surgeons who saw such cases would find the operation very useful—at all events to relieve the patient, if it did not cure the case. It would also teach others how to live in the tropics, and especially to avoid beer and whisky at lunch and dinner, and the enormous value of taking a sufficient quantity of plain water at proper times.

Mr. W. G. SPENCER said he was a learner from Mr. Keetley as to the performance of appendicostomy, but he asked that gentleman to state more definitely the underlying propositions which he assumed. Mr. Keetley did not note an objection, apart from the case, to the fixation of the cæcum by means of the appendix. The author omitted from his diagram the fact that by so doing he brought the ileo-cæcal valve close to the wall, and by so much fixed it. That might not be an objection in many of the cases, but it became an objection when he was going to extend the use of appendicostomy very largely, according to his present paper. He also hoped Mr. Keetley would say definitely what was the function of the appendix. The author put forward Professor Macewen on the one hand and Professor Metchnikoff on the other. In Macewen's original paper, what did he show as to the function of the appendix apart from the cæcum? And what evidence had Mr. Keetley to show that the appendix had any other functions than a corresponding area of cæcum? He supposed the author would admit that a similar area of cæcum could not be missed. From the developmental point of view it was nothing more than the shrunken tip of the cæcum, or a remnant. And, from the histological point of view, how could Mr. Keetley assume that the appendix was like a small pancreas or other organ? There was no reason to remove it if it was healthy, and therefore the Americans might be all wrong on that matter, but as a remnant in which there was retention it came under the same heading as a thyro-glossal duct, or the male breast, or the parovarium, or any other remnant in the body. They should not be removed by the surgeon unless there was good reason, but if retention occurred in them the question became surgical, and removal must be considered.

Dr. HERTZ agreed with Mr. Spencer that Professor Macewen's observations did not prove that the appendix was worth preserving. The mere fact that it produced a secretion was of little importance; nothing definite was known about its functions, but probably it was the same as that of the secretion of the cæcum and the rest of the colon. Mr. Keetley said that appendicostomy was the best surgical treatment of constipation. He agreed with the author that it was better treatment than the excision of the colon, but it was doubtful whether an operation was ever really necessary, and whether there were any cases of constipation which could not be cured by other means. He (Dr. Hertz) had observed by means of the X-rays that the majority of severe cases of constipation showed no delay in the passage of fæces through the greater part of the colon, the delay being confined to the pelvic colon and the rectum. It was not a question of sluggishness of the colon in these cases, but of inability to defæcate properly, owing to various causes, such as acquired anæsthesia of the rectal mucous membrane, weak abdominal muscles, and an atonic condition of the musculature of the pelvic colon and rectum. There were, however, some cases of severe constipation in which there was delay in the passage through the colon, but these were comparatively rare. He had not seen a case of constipation in which there was definite delay in the passage of the contents before the hepatic flexure was reached. Therefore he could not see the advantage of washing out

the colon from the appendix instead of from the rectum. By means of the X-rays he had seen, after injecting water containing a bismuth salt under low pressure *per rectum*, that the shadow of the cæcum appeared, which proved that it was easy to run water in through the rectum without great pressure as far as the cæcum. Usually only the lower part of the colon was involved, so that it was easier to wash it out from below, as the distance was shorter, than from the appendix to the seat of the faecal accumulation. In the milder cases of constipation cured by the operation, *i.e.*, those associated with appendicitis, he thought that the relief of the appendicitis and not the appendicostomy cured it. It was in the experience of most surgeons that the removal of a chronically diseased appendix might cure constipation, because the constipation was secondary to the appendicitis. This was analogous to the fact that in women inflammation of the pelvic viscera gave rise reflexly to constipation, and relief of the chronic inflammation cured the constipation. In a similar way appendicostomy would cure muco-membranous colitis in the few cases in which it was secondary to constipation due to chronic appendicitis. In most cases of muco-membranous colitis the lower part of the colon was mainly involved, so that washing from below, according to the method employed in Plombières and Harrogate, did as much good as from above. He thought it would be unjustifiable to do appendicostomy on a patient with typhoid fever, because it would be impossible to treat the ulcers in the lower end of the ileum as well as those in the cæcum and ascending colon. It would be very dangerous to pass a catheter through the ileo-cæcal valve where there was active ulceration of the ileum, and he thought such treatment unnecessary, because it was comparatively rare for the danger in typhoid fever to be produced by the ulcers. Perforation and hæmorrhage could not be prevented by local treatment with an appendicostomy. The main danger was the toxæmia occurring in typhoid fever, and that could not be avoided by the operation, as it originated from the bacteria actually present in the walls of the ulcers. He did not think the case of supposed intestinal toxæmia quoted by the author showed that the operation was of any value, because the patient was already almost well before the washing out was begun. The subsequent improvement might therefore have been due to the natural course of the illness. He had not been much impressed by the results obtained in Mr. Keetley's cases in which the operation was done simply to give nourishment, and, moreover, there was no difficulty about giving nutrient enemata or large quantities of saline solution *per rectum* if given slowly enough; 9 oz. of a nutrient enema could be injected at a time and retained, and many surgeons now used continual saline injections *per rectum* after abdominal operations and found no difficulty in carrying out the treatment. He thought appendicostomy was very valuable, and probably the best known treatment in severe ulcerative colitis, especially where the symptoms pointed to the first part of the large intestine being involved.

Mr. R. P. ROWLANDS expressed gratitude to the author for his paper, but thought that his enthusiasm was partly that of a lost cause, because the appendix could not be shown to be very valuable. For intussusception it could

not be of great value except where the cæcum was a very small part of the intussusception. He agreed that the delay in doing appendicostomy in a child with intussusception would be a serious danger to its life. With regard to appendicitis, it seemed very much better to remove the appendix, because to leave a persistent sinus and appendix in the abdominal wall in an inflamed and sloughing condition must be a source of danger, because suppuration in the planes of the abdominal wall might occur. Even after opening appendicular abscesses, cellulitis of the abdominal wall had been known to lead to death. It did not seem worth while to run these risks on the off-chance of the appendix being useful later in life. With regard to whether a transplanted appendix could be made use of years later, he would be afraid that the appendix so placed would atrophy so much that it would be of little future use. A serious risk in transplanting the appendix would be hernia. All the operations for appendicitis at present were designed to prevent hernia; even when there was an abscess a valvular operation was done, and to deliberately leave a guide for a hernial protrusion to follow was bad surgery. He agreed that treatment by dieting and by copious injections from the anus was far superior to treatment by appendicostomy, which could only be required in a very few cases of constipation. Moreover, many cases of constipation had got well in other ways. In these neurotic patients even a simple exploratory operation was sometimes followed by marked improvement. Of two cases in which he had explored, in one the patient was supposed to have a kinked hepatic flexure of the colon following nephrorrhaphy two years earlier. He found nothing abnormal, although he examined the stomach and all the intestines. The result was that from previously having constipation of a very severe kind, the patient afterwards had the daily use of her bowels, and that had continued for the last two years. In the other case, in which a growth of the intestine had been diagnosed, with which he concurred, he explored and there was no obstruction. A similar result happened there, the condition having been largely due to starvation, the dieting having been suggested by the supposed growth in the intestine. If appendicostomy was going to be done haphazard by surgeons, he believed there would be more cases of fistulae. It must be a very rare event to need the appendix for nutrition, for it would always be possible to feed the patient either by the stomach, or through the rectum, or subcutaneously. He agreed that appendicostomy was a very valuable operation for many cases of colitis. For the relief of intestinal obstruction he did not consider that even a well-dilated appendix provided sufficiently for drainage. In one of his cases of carcinomatous stricture of the ascending colon the ileum had perforated in spite of appendicostomy, which had been performed for acute, following upon chronic, intestinal obstruction.

Mr. H. F. WATERHOUSE said he felt very strongly that the Section owed a great debt of gratitude to Mr. Keetley. Personally, he did, because it was entirely to his writings that he (Mr. Waterhouse) had had his experience of appendicostomy. Though that had been limited to five cases, it had been so favourable as to warrant him in reporting it. So far he had only done that

operation for colitis, and he believed that in the future it would be considered the best form of treatment for colitis. Three of his cases were ulcerative colitis, and the operation had been for them absolutely curative. His experience of that condition before Mr. Keetley's papers appeared had been most unfortunate, and an operation which gave for such a resistant affection three successive cures within three weeks of the operation must be regarded as a great advance in surgical treatment. Two of his cases were mucous colitis; he admitted neither of them was cured, but in both cases there was considerable improvement. Both patients still passed a certain amount of mucus and they had some abdominal discomfort, but so much better were they that they expressed their willingness to go through the operation again if it were necessary. He could scarcely see Mr. Keetley's point with regard to the treatment of intussusception, and he would like to hear a fuller explanation of the action of appendicostomy in that condition; also would he say what amount of fluid he used for irrigation? He had been surprised to find that many intestines held 3 pints to 5 pints of normal saline without discomfort, and the patient did not seem to be in any hurry to expel it. With regard to the treatment of acute appendicitis, he could not help feeling very strongly, in spite of Mr. Keetley's arguments, that if he had appendicitis he would like his appendix removed. The removal of it was quite a satisfactory matter, and when once an inflammation was started in the appendix it was difficult to foretell what would happen during the next day or two. He agreed with a previous speaker that if the suppurating appendix were left it might start phlegmonous abscess or suppuration in the planes of the abdominal wall, and there was already sufficient trouble on that account after operating for acute appendix abscess. Subject to what he had said regarding colitis, he did not suppose the operation had a prospect of anything like the wide application which Mr. Keetley seemed to think it would have.

Mr. KEETLEY, in reply, thanked all who had shown such a kindly interest in his paper. But he could not believe that the operation was such a bad one as had been depicted. If all that had been said against it were true, he could not imagine a worse operation in surgery. Yet it had been done by many well-known surgeons, whom all respected. He had just heard from Sir William Macewen that he had done the operation in four of the indications mentioned in his (Mr. Keetley's) paper "with very excellent results." It was said by one speaker that suppuration in the abdominal wall was dangerous. True, and so was suppuration elsewhere. But the question was whether suppuration arising around the transplanted appendix was comparable with suppuration in the peritoneal cavity, when in the latter case the suppuration arose with the appendix still in the peritoneum or, as sometimes happened, after it had been placed in a bottle. He could not help taking notice of the number of positive statements which had been made without any experience being quoted in support of them. He had himself been careful about making positive statements, but when he did he brought forward a case or two. For example, when he said that an appendix placed in the abdominal wall was

comparatively safe, he quoted two cases, in one of which the appendix perforated spontaneously and in the other by fish-bones, and in both the symptoms were very trivial. Of course the appendix would sometimes atrophy with age, but why should it atrophy any faster in the abdominal wall than in the peritoneal cavity? In one of the cases the appendix was placed in the abdominal wall nearly a year ago, and only the other day was appendicostomy wanted, and then it was quite easy to do it. There was said to be a danger of hernia. Why? Hernia had not occurred in one of the cases. If the parts were properly knit together, and no superfluously large hole was made, why should there be hernia? He asked those who made that criticism whether they were not a little prejudiced against the operation. It was said that constipation was as well treated by enemata *per rectum*; but his cases showed plainly that it was not. It was not justifiable for anyone to make such a round statement with such cases staring him in the face. In Case 27 the patient was watched by his attendants. For three months she was not seen to pass a motion, yet she received *per rectum* enough water to float the *Dreadnought*. Immediately after appendicostomy an enema was administered by the appendix, and she passed a motion which bystanders said resembled the birth of a child, and it made almost as much commotion. It had also been said there was danger of a fæcal fistula after appendicostomy. How could that be unless the appendix sloughed? As to salines being capable of being given satisfactorily by the rectum, he said so in his paper. The class of cases in which he doubted the necessity, if not the efficacy, of appendicostomy was the class in which existed conditions of great depression and exhaustion. It had been said positively that the large intestine could be washed out just as well *per rectum* as with the aid of appendicostomy. If he were going to make a positive statement like that at the reading of a paper, he would ask himself if he was not prejudiced. Any long tube like the colon could be washed out better if it was open at both ends. If there was a hole in the cæcum, and if it persisted for a few days, it would be found generally when giving an enema that the water would quickly pass out through the hole; but often it would be some days before that was observed. Mr. Spencer naturally and sensibly criticized the opinion that the appendix had a valuable function. He knew there was much to justify doubt about that. The only positive observations were those of Macewen, but they were of great value and significance. He noticed in a case in which the cæcum lay open that when the contents of the small intestine flowed through the ileo-cæcal valve, there was at once a gush of thick alkaline mucus from the appendix. Perhaps there was some also from the cæcum, but the amount of mucus secreted by the appendix was greater. He protested against assertions as to the functions of an organ based upon its resemblance to some neighbouring part. Mr. Spencer mentioned the breast. That organ was a gigantic compound skin gland; but how different in function it was from the neighbouring axillary sweat glands! One might take an ordinary sweat gland or an ordinary intestinal gland and argue that it could not have a special function because it was only lined by epithelium similar to that of the neighbouring parts. The

function of the human body was marvellously more differentiated than was its recognizable anatomy. Nearly every abdominal surgeon knew that the delay of the fæces occurred mainly in the sigmoid and rectum, independent of what the X-rays had shown. And they did not show that the lodgment of fæces in the rectum and sigmoid was uninfluenced by what went on in the upper part of the colon. One of the best-known causes of constipation was not anything occurring in the large intestine at all, but stricture of the pylorus. One might as well say that the latter could not cause constipation because the X-rays showed in a certain number of constipated people that the fæces were collected in the rectum. The bad results spoken of by Mr. Mummery were, he thought, chiefly in the nature of sloughing. He (Mr. Keetley) had dealt with that in his paper. Though that was a danger, it could well be avoided by proper technique. Mr. Mummery also spoke about closing an appendicostomy; but if the operation was properly done no operation to close was needed; if left alone the opening would contract, sometimes rather too quickly. He had omitted to read the section of his paper describing the technique of the operation because he was addressing an audience of practical surgeons, and had barely time to read the rest of the paper. He had described how to reopen an appendicostomy which had been contracted for a week or two. Whoever operated on cases where there was known to be constipation was bound to find fairly frequently the transverse colon hanging low, and the cæcum in the pelvis, at the bottom of Douglas's pouch. Appendicostomy prevented the cæcum from slipping back into the pelvis again, and he believed that had something to do with its value in the treatment of constipation. Mr. Bidwell said the operation was of no use in intussusception, but how did he know? The cases narrated were not numerous enough to prove much in themselves, but nothing except demonstration by experience would make him believe that severe intussusception involving the cæcum, as it generally did, would take place as easily after appendicostomy had fixed the cæcum as before. Fixation of the intestine affected had been practised without appendicostomy for intussusception, and what had been wanting was sufficient experience to justify positive statements one way or the other. The time taken by appendicostomy for this purpose was very small. When a person had an acute illness, that was not the time Mr. Keetley chose for doing an elaborate operation. He simply buttonholed the abdominal wall and slipped the appendix through, fixed it, and left it. There was in this series of cases one of gangrene and perforation of the jejunum in which he had accused himself of wasting time through performing a useless appendicostomy; but the time was spent in getting to and examining the appendix from a median incision, owing to a mistaken diagnosis, and not in performing the appendicostomy. Sometimes mucous colitis was apparently secondary to appendicitis, but not as a rule. Anyway, both were cured by appendicostomy. With regard to its being better to remove the appendix because then there was no fear of recurrence, that remark had already been answered in the paper. Further, an appendix transplanted meant comparative freedom from risks which followed appendicectomy, when the site of the

appendix root was left in the peritoneal cavity to adhere to small intestine, &c. Mr. Bidwell also urged against the operation that it left a sinus; his answer was that an appendicostomy was a valvular opening lined by mucous membrane, and not a sinus. It was appendicectomy which often left sinuses. A month ago an old patient came to show herself, and Mr. Keetley, thinking it was one of his appendicostomy cases, said he could not understand why she showed so much discharge. The operation was done some months before; what could be the reason? On looking up the notes of the case, he found it was not an appendicostomy but a case of appendicectomy after abscess. In conclusion, he said he hoped he had not replied with any bitterness; he had felt greatly flattered and pleased by the amount of attention paid to his paper. Some of his views might be mistaken; he was sure some of those of his critics were; and he must leave the debated questions to be settled by time.

Surgical Section.

December 8, 1908.

Mr. J. WARRINGTON HAWARD, President of the Section, in the Chair.

A System of X-ray Examination of the Urinary Tract.

By W. IRNSIDE BRUCE, M.D.

THE methods of carrying out an X-ray examination of the urinary tract differ in many respects, and there can be no doubt that in the hands of each originator his own method yields the best results, but in making such examinations it is, in my opinion, absolutely necessary that some definite system should be followed. Having employed for some time, both at Charing Cross Hospital and St. Peter's Hospital, a system which has proved satisfactory, but which, nevertheless, I am sure is capable of further improvement, it has occurred to me that the method employed at these institutions might be of interest to you. I propose, therefore, without entering into unnecessary technical details, to indicate to you the essentials of the method I employ, and to show you some of the results.

Taking the X-ray as produced and looking upon it as a source of light emanating from a point afterwards referred to as the anode of the tube, the radiogram is a shadow cast upon a photographic plate of those parts of the body that are opaque to the X-ray.

With ordinary light, shadows may give very false impressions of the objects which cast them, and in the case of X-ray shadows of the renal region this fact claims specially to be borne in mind. If these X-ray shadows are to be used as a means of recognizing any existing abnormality it is surely necessary as a preliminary that an accurate knowledge of the normal X-ray appearance presented by this part should be acquired. In order to gain this knowledge it is of the greatest importance that the shadow secured of the part should be always the same—that is, the anode of the tube should always bear the same

relation to the part at the time of irradiation. The method I employ allows of the anode of the tube being placed readily and easily in accurate anatomical relation to the part to be examined by means of a very simple mechanical contrivance, and in dealing with the renal region it is placed immediately below the spine on the second lumbar vertebra. In every case this relation between the anode of the tube and the spine of the second lumbar vertebra is maintained, and, since the kidneys and the ureters usually bear a definite relationship to the bones of the part, a radiogram showing up these structures would demonstrate the shadow relationship they bear to the bones. To obtain a certain knowledge of these relationships the pelvis of the kidneys and the ureter of a post-

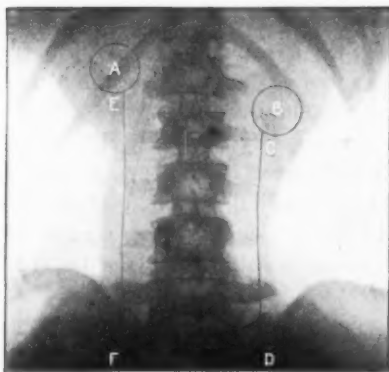


FIG. 1.



FIG. 2.

mortem subject were injected with an emulsion of bismuth, which is opaque to the ray. A radiogram was then secured of the renal region of the subject, the anode of the tube being in the usual relation to the second lumbar vertebra. By means of this radiogram the shadow relationship of the kidneys, right and left, and of the ureters was worked out, and in the radiogram (fig. 1) that relationship is distinctly indicated. The circles A and B = the pelvis of the kidneys and the lines C, D and E, F = line of the ureters.

The position of the subject is also of some importance. The patient is placed, lying face downwards with his arms by his sides, on a canvas-

topped couch under which the tube is arranged, and in order to restrict, so far as it is possible to do so, the movements of the diaphragm and consequently of the kidneys, a pad or compressor is placed under the abdomen with the whole weight of the patient resting upon it. The compressor pushes the abdominal contents away from the renal area and allows the more perfect illumination of these parts. The position of the patient on the couch is as to be seen in the photograph (fig. 2).

It is often difficult to secure a radiogram of good quality, sometimes on account of the density of the subject and sometimes owing to the vagaries of the patient. The latter is usually overcome by frequent examinations or by administration of an anæsthetic. With regard to



FIG. 3

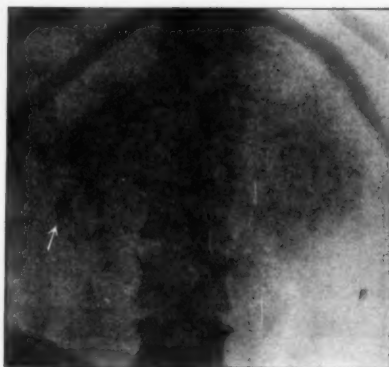


FIG. 4.

the first-named cause, it is not always the most obese subject that is the cause of failure; more often it is the stout subject with considerable muscular development. The use of an abdominal compressor, combined with frequent examination and effective evacuations of the intestine, usually results, however, in a radiogram of good quality, even in the most obese subject. A radiogram of good quality should show the following points: It should be absolutely symmetrical; the lumbar vertebra should be sharp and distinct in outline; the margin of the psoas muscle easily made out; the twelfth rib well defined; and the outline of the kidney on either side discernible. Fig. 3 is a radiogram showing all these points. If such a radiogram as this be secured there

is no reason that I can see why a positive or negative diagnosis may not be absolutely relied upon; but in a case where the radiogram is not up to this standard it is the duty of the radiographer to state this fact, so that the diagnosis, positive or negative, may be valued at its true worth.

Let me impress upon you that in order to bring renal examinations to a successful conclusion it is essential that the examination should not be carried out in a hurried manner, for not only is it necessary to have the intestine properly emptied, but time should be allowed for re-examination, not once, but two or three times if necessary. For example, the radiogram (fig. 4) is one of a case in point; the subject was not obese, but a man of exceptionally good muscular development. He came



FIG. 5.



FIG. 6

up for X-ray examination on two occasions, on neither of which was the abdomen well emptied. The radiograms were not of first-class quality and the result was negative. Finally he was admitted into the hospital, the bowels thoroughly evacuated, and irradiation carried out for the third time. An opacity marked → representing a small renal calculus is discernible. Also, the radiograms (figs. 5 and 6) of the case which was under the care of Mr. Stanley Boyd, where re-examination not only verified the presence of any opacity in the line of the ureter, but made the diagnosis more certainly correct in a rather interesting way. It happened between the examinations that the patient had an attack of colic, which resulted in the opacity representing the calculus appearing somewhat lower down in the line of the ureter in the second

radiogram. This observation is made possible by the radiograms on each occasion being secured in fixed position. The opacity marked → representing the calculus lies in relation to the transverse process of the fifth lumbar vertebræ, considerably nearer the crest of the ilium in fig. 6.

It is often insisted on that the radiographer be in possession of all the clinical facts of the case before making his examination. Although a knowledge of the clinical history is desirable, it is, however, better, in my opinion, that at the time of examination the only knowledge one should have of the case is that it be one of supposed calculus in the kidney, bladder or ureter. By thus approaching the case the



FIG. 7.

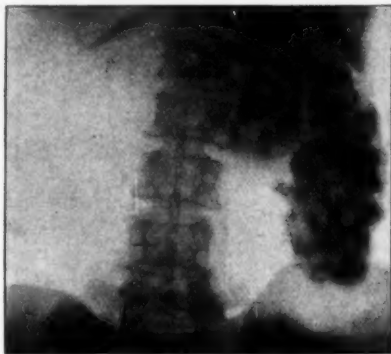


FIG. 8.

radiogram can be viewed with an entirely unbiased mind, and an examination in every case of the whole of the urinary tract becomes absolutely necessary. A provisional diagnosis having been made radiographically, the clinician can then compare his conclusion with that arrived at by the X-ray examination. Thus the value of clinical observation is considerably increased. Further, it is of the utmost importance that neither the clinical nor the X-ray evidence should be put in a place of first importance. The radiographer is wrong who states that X-ray evidence is infallible, and, on the other hand, the clinician is surely equally wrong in advocating operation in the face of strong X-ray evidence to the contrary. It is always advisable, if possible, to bring the one in line with the other. Many cases of positive X-ray

may be negative clinically and vice versa. For example, in a case in which, after a typical attack of renal colic, the pain subsiding and the urine becoming normal, the calculus having presumably passed, on X-ray examination (fig. 7) an opacity marked → was to be seen in the line of the ureter, and a calculus was removed by operation. As I have stated, no abnormality was to be found in the urine, and the only symptom was persistent slight pain. In this case the calculus probably completely occluded the ureter, thus preventing any abnormal urine reaching the bladder. In contradistinction to this case let me mention another, which was under the care of Mr. Gibbs. The patient suffered from repeated attacks of typical renal colic with sickness and hæmaturia; X-ray examination gave a negative result on three occasions.

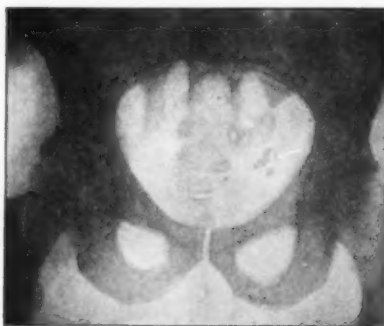


FIG. 9.



FIG. 10.

It was observed that the outline of the kidney on the affected side was much increased in size, so as to almost completely fill the right flank. Operation revealed a tuberculous kidney.

The radiogram (fig. 8) is that of another case in which there was no symptom other than the pus in the urine—no renal colic, no sickness, no pain in the back, yet no fewer than 284 stones were removed from the kidney by Mr. Waterhouse. On the other hand, let me give you an account of a case under the care of Dr. Baker and Mr. Pardoe, who have kindly supplied me with the clinical notes: Mr. P., aged 31. For two years seven or eight attacks of definite right renal colic, never any hæmaturia; has passed at least twenty or thirty small pieces of calculus; urine—no albumin, no pus, no bacteria. Plenty of calcium oxalate crystals. Most of the calculi passed were smooth and brown like

linseed, two were white and angular; calculi were last passed twelve months ago. Has had three or four attacks of severe colic since that date, but no hæmaturia. April 29, 1908: Right kidney palpable and tender, the left could not be felt; nothing to be felt *per rectum* or by bimanual examination except a little tenderness in both iliac fossæ; no increased frequency of micturition. X-ray examination (fig. 9) revealed an opacity marked → in the line of the right ureter, typical in shape and strongly suggesting the presence of an impacted calculus. May 7, 1908: Cystoscopy, bladder normal, both ureteric orifices normal, and a clear efflux on both sides; ureteric catheter passed without obstruction to the pelvis of the kidney on both sides. The patient, acting on the advice given him, went to Contrexéville. No calculus was passed, and, after returning, he had some attacks of renal pain.



FIG. 11.

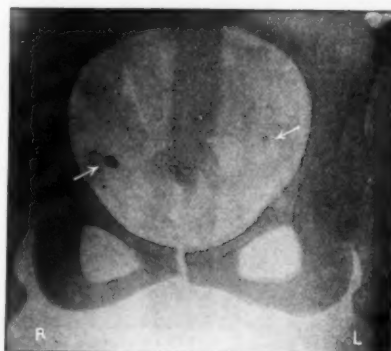


FIG. 12.

And yet another case, in which the usual examination (not cystoscopically) for the diagnosis of a vesical calculus was carried out with general anæsthesia, the result being negative. X-ray plainly shows a stone marked → in the bladder (fig. 10).

These cases, and many others I could show you, prove that in the diagnosis of renal calculus and other calculi in the urinary tract, both X-ray and clinical evidence must be taken into consideration. It often happens, however, that the evidence to be found in the radiogram is so strongly in favour of the presence of calculus that it can be said to be sound and reliable, even in the face of very weak or even no clinical support. Fig. 11 is the radiogram of such a case.

In the interpretation of opacities observed in the renal region, the radiographer has to face certain difficulties apart from mere technical faults, such as photographic stains, &c., but with experience and the assistance of many valuable writings on the subject, it is quite possible for him to avoid most of these difficulties. Sometimes, however, X-ray examination seems to render a case more obscure: to illustrate this point, let me give you shortly an account of a case under the care of Dr. Gibson and Mr. Pardoe, who kindly supplied me with their clinical notes: Mrs. B., a healthy young woman, six weeks after her confinement had a sudden attack of severe pain in the right loin, extending to the groin and vulva, accompanied by strong and indeed

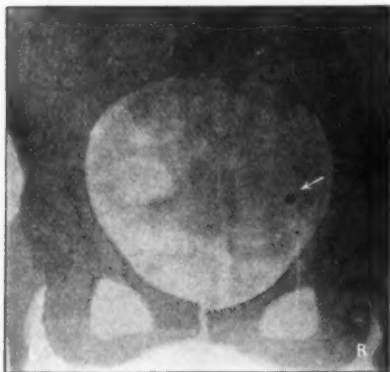


FIG. 13.



FIG. 14.

imperative desire to micturate. There was observed definite bright hæmaturia which lasted a few days; a second attack similar in nature came on about three months later and a third attack within a month after. When seen by Mr. Pardoe (October 2), the following was her condition: there was no cystitis, only a trace of albumin in the urine, no blood, and a few hydatid casts; the right kidney was just palpable on deep inspiration, the left kidney could not be felt; nothing to be felt on bimanual palpation, abdominal or vaginal; cystoscopy showed a perfectly healthy bladder and ureteric orifices, and a clear rapid efflux on both sides. Patient complained of a steady, aching pain in the right groin and loin whenever she was up and about; this disappeared entirely in the recumbent position. The result of the X-ray examination,

carried out on October 10, is to be seen in Fig. 12. Two distinct opacities marked → are to be made out, one on the left side, the other on the right, each in line with the ureter, and strongly suggesting a calculus impacted in the lower part of each ureter. Seeing that the calculi were small enough to pass, the patient was advised to take copious diuretics and to keep a careful watch on the urine. Further history from Dr. Gibson (November 10): Two weeks after the examination, the patient going about and taking plenty of liquid, developed, on November 8, severe renal colic on the *left* side with slight hæmaturia. No stone was observed nor felt to pass, the watch on the urine having been relaxed. Fig. 13 is the result of an examination carried out on December 7, 1908. You notice that the opacity on the left side has disappeared,

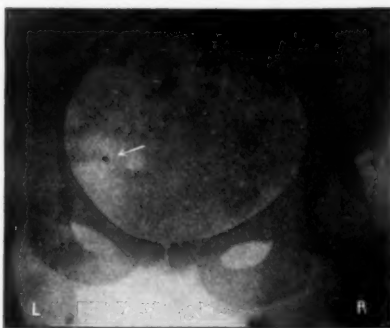


FIG. 15.

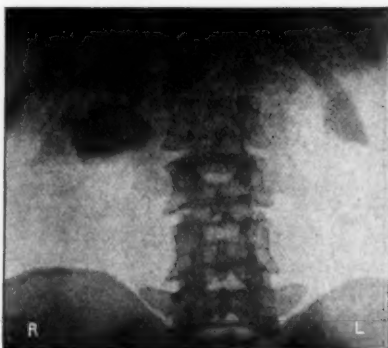


FIG. 16.

the calculus in left ureter, observed on first examination, having been passed *per urethram*.

In the system of examination of the urinary tract described, both kidneys and ureters, right and left, are examined at the same time. The following cases illustrate the importance of investigating both sides, irrespective of clinical indications:—

(1) A case in which the symptoms of renal calculus were complained of on the right side, and the right side only, you can see in the radiogram, an opacity marked → strongly suggesting a calculus in the pelvis of the *left* kidney (fig. 14).

(2) A case in which severe pain was complained of in the right groin, an opacity marked → is plainly discernible near the lower end of the left

ureter. In this case, subsequent to this discovery, pain was complained of on the *left* side, and eventually a small calculus was passed (fig. 15).

(3) Another case is one in which the explanation of the presence of pus in the urine was sought for by X-ray examination of the renal region, other sources having been excluded. In the radiogram there is to be seen ample evidence in either the right or the left kidney of the source of the pyuria. Should only one side have been examined, sufficient evidence would have been procured to have suggested operative interference, a measure which, in view of the condition of the other kidney, would, I take it, have been contraindicated (fig. 16).

The radiographer can often do more than indicate that the case is one of calculus of the kidney or ureter. Any increase in size of outline



FIG. 17.

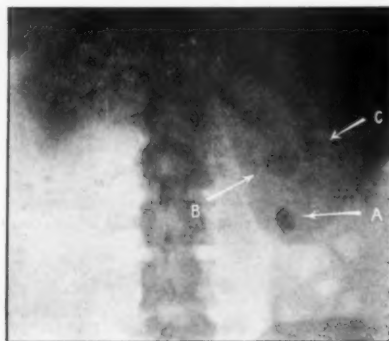


FIG. 18.

of the kidney, when observed in a case of suspected renal calculus, may assist the diagnosis. For example, the radiogram (fig. 17) is that of a boy who suffered from attacks of renal colic associated with pus in the urine. Calculus was suspected, but on X-ray examination the outline of the right kidney was markedly increased in size; no opacity suggesting the presence of calculus. From an X-ray point of view, the diagnosis in this case, in view of the increase in size of the outline of the kidney, is tuberculous kidney, with renal colic from the passage of pus and debris through the ureter. As a matter of fact, such was the condition of affairs on post-mortem examination.

If, moreover, there is more than one calculus present this fact can be noted, and the position of the calculus or calculi, in relation to the pelvis or the cortex of the kidney, may be indicated. The condition of the sound kidney, provided the outline of it can be made out, may also be deduced from the size of the shadow cast by it. The following cases illustrate these points.

(1) A case in which it is obviously possible to count the stones in the kidney and to indicate, at any rate roughly, their position in relation to the kidney—namely, three stones, one in the lower pole of the kidney, marked \rightarrow A, one in the pelvis of the kidney, marked \rightarrow B, and one impacted in the calices, marked \rightarrow C, about the centre of the pelvis (fig. 18).

(2) A case of obvious stone in the cortex of the kidney, where the outline of the unaffected kidney is plainly to be seen, slightly increased in size (fig. 3).

In conclusion, I trust that you will appreciate some of the difficulties the radiographer has to meet in connection with the diagnosis of renal calculus, and you will see that he has striven to some purpose to remove this method of investigation from the realm of mere mechanics to a branch of the profession that must be recognized as a permanent and essential factor in the accurate diagnosis of disease of the urinary system.

DISCUSSION.

The PRESIDENT (Mr. Warrington Haward) remarked that it would be agreed by all present that a very beautiful series of radiograms had been demonstrated by Dr. Bruce. It would also be conceded that it was desirable not only that radiograms should be good, but that medical men should be familiar with their proper interpretation. From time to time one had seen curious errors made with good radiograms because of the unfamiliarity of the observer with the natural appearance presented by the parts depicted. Therefore it was a great advantage to have so lucid a demonstration as that furnished by Dr. Bruce of a part of the body wherein diagnosis might be materially assisted or even rendered certain by X-ray examination. Members of the Electro-Therapeutical Section had also been invited to be present, and he was sure their views would be welcome. He asked if any Fellows could tell the Section of cases in which difficulty had arisen as to the nature of the shadow, for instance, phleboliths being mistaken for calculi. It was always interesting to hear of errors, if it

was not always pleasant to relate them, as it might save others from the same pitfalls.

Dr. HARRISON ORTON said he had been much interested in Dr. Bruce's paper, and thought he had clearly shown that the X-ray examination of the urinary tract was not the simple mechanical process it was by so many thought to be, and that the inexperienced interpretation of negatives, which often lacked the proper qualities, had been and was still responsible for many of the errors attributed to this method. With regard to technique, that which he employed at St. Mary's Hospital and the Royal Free Hospital was so similar to that employed by Dr. Bruce that he would not say much about it, the only difference being that he used some compression in addition to the weight of the patient by screwing down a board on to the plate placed on the back of the patient. He attached a great deal of importance to the quality of the rays emitted by the tube at the time of examination; they should be of such a quality that they did not penetrate the least dense calculi. He need hardly enter into the technique of this before the Section, but they would readily understand that many calculi might be missed by employing a ray which had sufficient penetrating power to pass through them. The difficulties which Dr. Bruce had mentioned of obtaining satisfactory negatives in some cases were well known to radiographers, but he agreed with him that in expert hands most of these difficulties could be overcome, though several examinations might be necessary. He found the tissues of very old people were sometimes very opaque, and he had had most trouble with an old lady aged 84. The recognition of the correct quality of negative required was an essential of technique, and if such a negative could not be obtained, a rarity now, as he had said, this fact must be stated, or a reliable negative diagnosis could not be given. He would like to emphasize the fact mentioned that a thorough evacuation of the intestines was necessary for many reasons, which he had stated in a previous paper read before the Society. He agreed that we should not be influenced by the clinical facts of the case, for the reason that he thought the whole urinary tract ought to be examined in every case, for large calculi might be present with practically no symptoms; symptoms might point to the wrong side; calculi might be present in both kidneys, or present in a kidney and ureter at the same time. It was important, however, that all clinical symptoms when present should be taken in conjunction with the X-ray findings, for, as he had pointed out both before this Society and at Sheffield this year, there were several conditions other than calculus which produced similar shadows, and which might lead to error if the X-ray findings were relied upon alone. Such were phleboliths in the uterine and prostatic veins, calcified mesenteric glands, concretions in the appendix, &c. He had prints of several of them which they might like to see afterwards, and also two phleboliths removed from one of the cases by operation. In conclusion, he would say that this X-ray examination, though not infallible, had in expert hands a percentage of error which was less than any other, and might, taken in conjunction with other methods, give information unobtainable by any other means.

Mr. W. G. SPENCER handed round the negative sent him by Dr. Ling of a patient aged 50, a man who for twenty years had supposed himself to be the subject of intermittent or cyclical albuminuria, and who began his medical attendance under the late Dr. Andrew, of St. Bartholomew's Hospital. The usual questions arose in that connexion in respect of a desire to insure his life, and after the matter had been debated the life was loaded. He was supposed to be frequently having attacks of albuminuria, and latterly he had wasted considerably and got weaker. He had had no particular symptoms, except that he could remember having sometimes had vague pains on the right side, until a sudden attack on the left side, which was at first thought to be true colic from some intestinal obstruction. But when, after a week, his bowels having been opened, there was remaining tenderness, a radiograph was taken. Before that was finished, however, he had a second attack, which was more definitely renal. The radiograph showed an opacity on the right side corresponding to the pelvis of the kidney. It was a large coral-shaped calculus with hydro-nephrosis above. What had brought things to a crisis was a calculus the size of the top of the little finger on the left side. The history of the case, shortly, was that in order to relieve the dangerous renal colic, which was threatening to recur, he operated on the left side and removed the stone corresponding to that opacity. It turned out that in the urine, when centrifugalized, what was thought to be albumin was pus. He therefore removed the whole of the right kidney with the large coral-shaped stone, and the patient had recovered. With regard to one of the slides shown by Dr. Bruce he suggested that the stone beside the ureter would, if left, develop into an encysted calculus of the bladder. That was a subject in illustration of which he had a patient before the Clinical Society two years ago, and Mr. Clutton, who was then President, had another. The discussion turned on the older subject of encysted calculus of the bladder. The stone was now pouching the lower end of the ureter above the sphincter and slipping down beside the mucous membrane of the bladder. If that was left alone, he thought there would be a pedunculated swelling into the bladder, covered with what had been regarded as a capsule, but was the mucous membrane of the bladder.

Dr. N. S. FINZI said he had listened with great interest to Dr. Bruce's lucid exposition, but he was sorry he had not touched on the subject of the diagnosis of other diseases of the urinary tract, such as tuberculous kidney, by means of X-rays. He had a case in which there was subsequently proved to be a caseous kidney. The shadow cast by this on the radiogram was deeper than that of any calculus; the density of the shadow was comparable with that of the bodies of the vertebræ. He thought the one criterion of a good X-ray negative should be a distinct outline of the kidney. He did not agree with those who said if the psoas muscle was seen it was enough. He had seen skiagrams in which no trace of a calculus was shown, but when one was taken indicating the kidney clearly, a stone was detected.

Dr. IRNSIDE BRUCE, in reply, agreed that the X-ray diagnosis of tuberculous kidney was a subject to which more attention ought to be paid. The caseous patches to be found in this disease cast shadows not easily distinguishable from those due to calculus. Opacities due to caseous patches could usually be recognized, however, by (1) the enlargement of the outline of the kidney shadow, indicating enlargement of that organ; (2) by such opacities usually showing a radiate arrangement; and (3) by the appearance of tuberculous calcified glands outside the renal area or even on the opposite side. With regard to the so-called phleboliths, their position in relation to the bones usually, in his opinion, served to distinguish them from opacities due to calculus. In the system that he had described the exact position of the ureters in relation to the bones was known, as were also the areas in which phleboliths were likely to occur. Unless radiograms are secured in a fixed position, the line pursued by the ureters is a mere matter for speculation. Phleboliths in radiograms secured in with the anode below the symphysis pubis lie well within the line of the ureter. He had been much interested in the explanation of one of the cases quoted, offered by Mr. Spencer, because it was consistent with the position of the opacity in the radiogram—namely, not more than $\frac{1}{2}$ in. from the lower end of the ureter. The calculus might well have become encysted in the bladder wall, and in that position allowing a free efflux and the free passage of a ureteric bougie, it would not occlude the ureteric orifice.

Surgical Section.

January 12, 1909.

MR. J. WARRINGTON HAWARD, President of the Section, in the Chair.

On the Functions of the Colon in Relation to Colic Exclusion.

By ERNEST W. HEY GROVES, M.S.

With the collaboration of I. WALKER HALL, M.D.

THE functional diseases of the colon and their relation to surgical treatment are at present the subjects of great diversity of opinion. This arises from the fact that, as in the case of all functional diseases, their pathology is somewhat obscure, and also that there exist all gradations between cases of purely functional and purely structural disease. But, in considering a disease which results from a perverted function rather than from a diseased structure, we may obviously hope to learn from physiology that about which morbid anatomy is silent. The employment of the operation of colic exclusion or ileosigmoidostomy for these functional diseases has been greeted in much the same way as all other surgical innovations. By a few it has been lauded as an almost infallible cure, by the majority it has been condemned or ignored as mere foolhardiness, and its ultimate place in accepted practice will depend upon the careful collection of facts and the ultimate results of operations. In the same way the operation of gastro-enterostomy has been regarded as a remedy for all stomach diseases, or condemned as useless except as a palliative measure, whereas now there has arisen a pretty common agreement among surgeons and physicians as to its utility and limitations. The object of the present communication is to bring forward some facts relating to the anatomy and physiology of the colon as derived from the study of three cases.

R. D., a single woman aged 38, came under my care at the Bristol General Hospital, where I first saw her in consultation with Dr. Michell Clarke, in September, 1906. She had had no definite previous illnesses, but for several years had been subjected to increasing constipation. At first purgatives and enemata relieved this, but latterly no drug taken by the mouth had any effect except that of producing colicky pains. During the time she was under Dr. Clarke's care the only treatment which secured a regular evacuation was the giving of a pint of olive oil high up the rectum each evening and allowing it to be retained, and the following of this by turpentine and soap and water the next morning. The abdomen was greatly distended, and I had never seen such distension before in the absence of definite obstruction. There was, however, no visible peristalsis, nor was there anything in physical signs to point to the nature of the disease. An exploratory operation was decided upon as her condition was such as to make her a permanent invalid. But the free exposure of the abdominal contents revealed nothing but a much dilated colon. This dilatation was such as to cause all the other viscera to be obscured by the ever-protruding coils of large bowel. There were no adhesions or any evidence of thickening or ulceration. I therefore cut the ileum across a few inches from its termination and sewed the proximal end into the side of the pelvic colon at its upper part, closing the distal end entirely. She made a speedy recovery from this operation, and its results were at first quite satisfactory. Up till the end of the year—that is, for three months—she was still rather inclined to be constipated, but this was easily relieved by an aloes pill or a simple enema. But in the beginning of 1907 she had an attack of diarrhoea with the passage of blood and mucus, and she continued to suffer from attacks of this kind for nine months. Dr. Clarke again kindly admitted her under his care in the spring of 1907. Her condition then was quite noteworthy. As a whole, the abdomen was no longer distended, but when the attacks of pain began, which they did almost daily, an irregular swelling occurred, which occupied the flanks and epigastrium, and was most conspicuous on the right side. This swelling was tympanitic to percussion and also rather tender. Her bowels acted four to six times daily, and the motions were very offensive, containing a little blood and a quantity of mucus. Her temperature was extremely irregular, rising at times to 102° F. or 104° F., but this rise of temperature was quite unaccompanied by any of the usual concomitants of pyrexia. When a nurse held the thermometer during the registration of the temperature it never rose above 100° F. On one

occasion, when the localized swelling was most prominent, we examined her under an anæsthetic, and as she became unconscious the abdomen assumed a perfectly normal appearance. We were inclined therefore to regard neurosis as the chief element in the case, and to treat the patient for a time with a little wholesome neglect. However, she continued to complain of constant abdominal pain and diarrhœa, and I admitted her to the Cossham Hospital in October, 1907. For three months she was kept under careful observation and medical treatment, but without much alteration occurring in the condition above described. On several occasions we had the diet carefully regulated and measured and all the fæces kept for analysis. The motions were copious, loose, and very offensive. In the first place an attempt was made to determine the rate of progress of food through the alimentary canal, as the most natural assumption is that after exclusion of the greater part of the colon the food will pass too rapidly through the digestive tract to allow of proper absorption; and it was found that sixteen hours elapsed between taking such things as currants or grapes before their debris could be recognized in the motions. This is not much different from the twenty-four hour interval which elapses in about one-third of normal individuals, according to Harley and Goodbody [4].

Date (1907)		Weight of fæces in grm.		Percentage of water in fæces		Percentage of solids in fæces
Dec. 11	...	115.4	...	86.14	...	13.86
" 12	...	65.7	...	74.28	..	25.72

WATER BALANCE.

Date (1907)		Intake		Output		Absorbed
Dec. 11	...	1560 c.c.	...	100.3 c.c.	...	1459.7 c.c.
" 12	...	1860 "	...	47.9 "	...	1812.1 "

Date (1907)	Calories	INTAKE			OUTPUT		
		Nitrogen in grm.	Fat in grm.	Carbohydrate in grm.	Nitrogen in grm.	Fat in grm.	
Dec. 11	697	5.86	36.70	128.40	1.39	3.22	
" 12	1040	6.86	21.57	197.96	1.25	3.61	

These results show that after a partial exclusion of the colon (1) the absorption of water is abundant, the diarrhœa being due simply to the discharge of mucus; (2) the amount of nitrogen and fat in the fæces, although high in proportion to the intake, represents only the almost irreducible minimum that is always present in the fæces. Thus it has been shown that adults pass about 8 grm. of bacteria in the fæces daily, and this amount varies from 2.6 grm. in constipation to 20 grm. in diarrhœa. Further, that the fasting individual passes 3.47 grm. of solid

material in the faeces daily [10], which includes about 0.57 gm. to 1.3 gm. of fat [13]. Examination with the sigmoidoscope revealed nothing abnormal. The same swelling recurred constantly in the position of the ascending and transverse colon, and the same fluctuating temperature was noted.

On January 24, 1908, I reopened the abdomen and excised the whole of the large gut from the stump of the ileum to within a few inches of the anastomosis. The piece removed was 29 in. in length and presented none of the remarkable dilatation which was so conspicuous on the first occasion. She made an uneventful recovery from this, and she has since been very greatly improved in health so that she has been able to resume her occupation as dressmaker. The motions are still rather loose and copious, but she is almost free from pain and has increased in weight from 6 st. 4 lb. to 7 st. 3½ lb. The analysis of the faeces on a recent occasion I append to this description of her case.

Date (1908)	Weight of faeces	Percentage of water in faeces	Percentage of solids in faeces
Oct. 8 ¹	390	96.16	3.84
" 9	213	89.68	10.22
" 18	149	84.64	15.36

WATER BALANCE.

Date (1908)	Intake	Output	Absorbed
Oct. 8 ¹	780 c.c.	275 c.c.	505 c.c.
" 9	1,050 "	189 "	871 "
" 18	480 "	126 "	254 "

Date (1908)	INTAKE				OUTPUT		
	Calories in gm.	Nitrogen in gm.	Fat in gm.	Carbohydrate in gm.	Nitrogen in gm.	Fat in gm.	Carbohydrate in gm.
Oct. 8	949	7.48	61.80	88.40	0.74	2.04	0.90
" 9	1,150	5.28	39.56	85.10	1.44	2.99	1.10
" 18	714	10.86	19.36	119.80	0.77	3.49	1.38

¹ Note.—On October 8, at the beginning of these observations, she suffered from some nervous diarrhoea, which accounts for the high proportion of water in the faeces.

In these figures two facts are evident: (1) That although an abnormally high proportion of water is passed in the faeces yet there is abundant absorption when plenty of fluid is taken; (2) that the absorption of nitrogen, fat, and carbohydrate is practically normal—that of nitrogen being on two occasions 90 per cent. and that of fat and carbohydrate being but little different from the normal average. There does not seem to be so much water absorbed after the colon has been removed as after its partial exclusion, and the improvement in health which follows the colectomy cannot be due either to an alteration in water or

food absorption. It probably results from the removal of a receptacle in which from the stagnation of its contents some toxic absorption takes place, and I will refer to this question again later on.

The above is clearly a case of idiopathic dilatation of the colon, and it is almost certain that it is not of the congenital variety. The first point to be noted is that the partial exclusion of the colon certainly cured the constipation—in fact, it led to the opposite condition. As to the nature of the dilatation, I can only surmise that it may have been due to a loss of tone and to some obscure nerve influence. The way in which it caused a phantom tumour was quite remarkable, but R. H. Fitz [3] has noted and discussed this relationship in 1899. The subsidence of the tumour under the anæsthetic is strongly suggestive of a nervous origin. But why the colon, when practically excluded from the digestive canal, should cause so much pain, and why its removal should produce so much relief, is not at all clear. But there certainly seems to be increasing evidence that this is so. Lowenstein [11], last year, in an exhaustive article, collected facts from no fewer than 132 different sources, and his references and bibliography practically make any detailed quoting of authorities on my part unnecessary. He comes to the conclusion that these cases require surgical treatment, and that the best form of treatment is excision of the large gut down to the pelvic colon. His table is as follows:—

			Treatment	
			Medical	Surgical
Number of cases	...	59	...	44
Died	...	39 (66 per cent.)	...	21 (48 per cent.)
Unaltered	...	4	...	2
Improved	...	9	...	7
Cured	...	7 (12 per cent.)	...	15 (34 per cent.)

Ito and Soyesima [6] relate twenty-one cases of resection, of which fourteen gave good results, five dying during the operation, one dying of diarrhœa, and one remained unchanged. And in this country Mr. Arbuthnot Lane [9] has described thirty-nine cases in which the colon was either excised or short-circuited. And his conclusion is that in the majority of cases requiring operation the mere ileosigmoidostomy is not sufficient, but an excision of the colon as far as the splenic flexure is necessary for complete relief. But when the heavy mortality of the operation is considered, Ito and Soyesima giving it at 29 per cent. and Lane at 23 per cent., it is clearly of the utmost importance to determine, as far as possible, the exact indications for its performance.

But before discussing this point any further I will give some details of my second case, which is of quite a different character, but from

which, with the co-operation of Professor Walker Hall, I have been able to obtain some further facts relating to the functions of the colon. F. B., a youth aged 19, was admitted to the General Hospital in a condition of profound collapse. He had been awakened at night, forty hours previously, by sudden abdominal pain and vomiting, which continued until admission. His previous history pointed to some indefinite "indigestion" and constipation. The pulse was 148, temperature 101° F., and respiration 36. The abdomen was distended, rigid, and tender, with free fluid in the flanks. On September 11, 1907, I opened it over the appendix and found much fæculent fluid in the peritoneum, with some solid fæces in the right iliac fossa. There was a large rent in the cæcum at the base of the appendix. The appendix was removed, the rupture sewn up, the peritoneal cavity mopped out with dry swabs and drained in the iliac fossa and the right loin. As there did not appear to be any adhesions limiting the peritoneal invasion we did not expect him to survive more than a few hours. He was immediately transfused continuously by the rectum, absorbing 12 pints of saline in twelve hours, and his recovery has been the most remarkable instance of the efficacy of this treatment that I have ever met. For forty-eight hours his condition varied between drowsy stupor and active delirium, and as he emerged from this he suffered from a severe degree of bronchitis, with some pleurisy, and I might remark incidentally that this tendency to lung complications is frequently noticed in cases of abdominal sepsis which have been treated by any form of transfusion. By September 20 the wound over the cæcum was discharging fæces, and it was evident that the suturing of the cæcum had broken down. On September 22 he had a furious secondary hæmorrhage, which would certainly have been fatal if it had not been for the promptitude of the house surgeon (Mr. Coulson), who held a mass of gauze in the wound until my arrival. On opening the wound the bleeding vessel was found at the brim of the pelvis, in an area in which the peritoneum seemed to be literally digested. The vessel was tied and the peritoneum sewn over it. The cæcal rupture was again sewn up, 3 pints of saline were injected into the median basilic vein, and the pulse became once more perceptible. His further progress was uneventful, except for the fact that the cæcum again and again opened into the wound, in spite of four operations at different intervals to close it. But on October 21 the bowels acted naturally, and continued to do so afterwards. Between this date and January, 1908, the opportunity was afforded of comparing the fæces coming through the cæcal fistula with those which were passed *per*

anum. I will speak of these observations later. On January 29, 1908, I performed an ileosigmoidostomy, uniting the ileum to the sigmoid laterally and then dividing the small gut about 3 in. from the cæcum. The ascending colon was too much buried in adhesions to permit of uniting the ileum to that part of the colon. During this operation a number of tubercles were observed upon the lower part of the ileum, and there were large glands in the mesentery. On February 2 the bowels were opened after an enema, and from that date onwards he has had a natural daily evacuation. On February 28 the fistula was closed in four layers, and this time healed without further relapse. He has remained in good health since and is now at work as an engineer. His weight, which was 6 st. 10 lb. in November, 1907, increased to 9 st. 6 lb. in October, 1908, and both scars are soundly healed. During the period between October, 1907, and January, 1908, he was able to take ordinary diet, and as a large fistula opened from the cæcum on to the abdomen it was possible to compare the composition of the fæces at the beginning and at the end of the large intestine, and so infer the changes that occur in that part of the gut. This was done on several occasions, and the most satisfactory means of collecting the fæces from the fistula was by tying in a Paul's tube, which remained in place for two or three days at a time.

ILEOCECAL FÆCES.

Date (1907)	Weight in grm.	Percentage of water	Percentage of solids
Oct. 30 ...	108.5 ...	94.74 ...	5.26 ...
" 31 ...	279.5 ...	90.5 ...	9.5 ...

RECTAL FÆCES.

Weight in grm.	Percentage of water	Percentage of solids
93.0 ...	89.6 ...	10.4 ...
51.2 ...	90.0 ...	10.0 ...

WATER BALANCE.

Date (1907)	Intake in cubic centimetres	Output in feces in cubic centimetres	Quantity absorbed in cubic centimetres
Oct. 30 ...	1,350 ...	205 ...	1,145 ...
" 31 ...	1,500 ...	113 ...	1,387 ...

ABSORPTION OF FOOD CONSTITUENTS.

Date (1907)	INTAKE				OUTPUT IN FÆCES					
	Calories	Nitrogen	Fat	Carbo-hydrate	Cæcal Nitrogen	Cæcal Fat	Rectal Nitrogen	Rectal Fat	Total Nitrogen	Total Fat
Oct. 30	2,689	14.18	159.4	280.8	0.34	1.1	0.62	1.53	0.97	1.64
" 31	2,576	12.0	159.4	268.1	0.29	2.2	0.66	0.28	0.95	2.4

The most striking thing about these results is the comparatively slight alteration that occurs in the fæces in their passage along the large bowel. There is, of course, an absorption of water, but this only amounts to about 5 per cent., and the absorption of fat or nitrogen is so small as to be negligible.

As regards the rate at which the contents passed through the two portions of gut, that in the small intestine was very constant, varying only between four and six hours. When he took a fig the seeds appeared in the fistula within four hours and continued to be discharged for about four hours more. The same result was obtained by carmine feeding. The time taken by particles in traversing the colon was more variable, but usually occupied sixteen to twenty hours. This latter result, of course, is related to the fact that this boy had a very regular daily action of the bowels, so that the greater part of the contents of the colon were passed as faeces on the following day. The disproportion between the time occupied by the colic contents in their passage and the slight changes they undergo therein is very noteworthy. But whilst this is true of the colon when filled with ordinary faeces as supplied by the small intestine, it does not seem to be the case when the empty colon is filled with fluid. It was observed very early in the case when administering enemata, that when more than a certain quantity was given, the fluid welled out from the fistula. This point seemed to be of some importance in consideration of the possibility of complete irrigation of the colon, and we therefore observed it with great care. When a coloured solution was used and poured into the rectum by a tube and funnel, when 15 oz. had been given the fluid appeared at the fistula, and this quantity never varied more than about 1 oz. When it appeared it did not do so in a steady stream, but in jets or waves which occurred four or five times a minute, clearly showing that the colon was undergoing slow rhythmic contractions. A solution of egg and milk to which some liquor pancreaticus had been added was injected into the rectum and some of the escaping fluid collected at the fistula. This was done after the colon had been thoroughly washed out. The result is seen in the following figures:—

	Original material injected		Fluid escaping from fistula	
Water	...	96.41 per cent.	...	89.68 per cent.
Solids	...	3.59 "	...	10.32 "
Nitrogen	...	0.38 "	...	1.82 "
Fat	...	0.73 "	...	2.0 "

From this it appears that in the empty colon the absorption of water is extremely rapid, the solution having lost over 6 per cent. in the few minutes occupied in passing from the anus to the fistula; but that the other constituents pass through without much absorption.

The last observations which we have made on this case relate to composition of his faeces now that the ileosigmoidostomy has been performed

and the intestinal contents pass from the small gut straight into the pelvic colon. The bowels act with the greatest possible regularity, at the rate of one motion each day. The motion is perfectly natural in colour, consistency and appearance.

Date	Total quantity of faeces	Percentage of water in faeces	Percentage of solids in faeces	Water in faeces
March 20, 1908 ...	40.6 gm.	79.8 ...	21.2 ...	32 c.c.
October 2, 1908 ...	54 "	75.93 ...	24.07 ...	41 "
" 5, " ...	49 "	69.24 ...	30.76 ...	34 "

Date	Nitrogen in faeces	Fat in faeces	Carbohydrate in faeces
March 20, 1908 ...	1.4 gm.	6.13 gm.	Not determined
October 2, " ...	0.75 "	2.49 "	"
" 5, " ...	0.78 "	4.92 "	1.512 gm."

Note.—During all these observations he was taking a mixed diet at home.

The results of the chemical examination of the faeces from the caecum and from the anus in the case of caecal fistula agree closely with those obtained by other observers [12 and 8] in showing that water absorption occurs in the colon, and a very slight absorption of nitrogen and fat. But as far as I am aware these are the first chemical observations on the metabolism after ileosigmoidostomy or removal of the colon. They show that water absorption is amply carried out by the rectum and pelvic colon after the great part of the large intestine has been partially excluded. And whilst the absorption is not so good when the colon has been removed, it is quite sufficient for the nutritive economy. The symptoms arising in the partially excluded colon which are relieved by its removal still require an explanation. This is found, I think, in the stagnation occurring in a dilated colon which has been shut out from the direct faecal stream, and in the decomposition and absorption of toxic products which accompany this stagnation. But there is a further factor of great importance which has hitherto been overlooked in this connexion. This is the occurrence of antiperistaltic movements in the colon and particularly in its ascending or transverse portions. Antiperistalsis has been shown to be the most predominant movement in the proximal parts of the colon of animals by Jacobi [7], Cannon [1], Elliott and Barclay Smith [2]. And each of the cases I have related demonstrate its occurrence in human beings. In the first case the dilated colon, after its partial exclusion, showed peristaltic waves of contraction, now in one direction and then in another. In the second case fluid could be observed escaping from the caecum in jets due to antiperistaltic contractions of the colon. Now, whilst this may not be of much significance in normal cases, it is clear that it may assume the

utmost importance when the colon has been cut off from the ileum and forms a large blind sac, and this will be particularly the case when it is dilated. In such conditions the antiperistaltic contractions may force back fluid, gas, and faeces into the blind gut and so cause pain, stagnation, and toxæmia, and I have lately met with a case which seems so exactly to prove this, that I venture to give the details.

H. W., a single lady aged 41, was under the care of Dr. Elwin Harris, through whose kindness I am permitted to relate her history. She was neurotic, sallow, emaciated, and weak, and had for seven years had such obstinate constipation that she never had a natural evacuation of the bowels. A right inguinal colotomy was performed, and within three weeks the colon was emptied of a large quantity of solid faeces by irrigations from both ends. Some months later the transverse colon became acutely inflamed (as proved by laparotomy), and after this the constipation and passage of mucus recommenced and irrigation of the large bowel was impossible. An ileosigmoidostomy, with complete division of the ileum, was therefore performed twelve months after the colotomy, and this at first acted very well, the patient having the first natural use of the bowels for eight years. But two months later faeces began to be discharged from the colotomy wound, and during a further period of four months more and more escaped by the colotomy and less and less by the anus, until at last nothing at all was evacuated by the rectum. She died twenty-four hours after the colon had been excised with symptoms of cerebral embolism.

In this case there is an absolute demonstration that in the partially excluded colon there exists so marked a reverse peristalsis that all the faeces were returned from the sigmoid to the ascending colon. If this is the explanation of the pain and general ill-nutrition after partial exclusion of the colon, it is evident that it must be met by removal of the colon, or else by providing for the drainage of its proximal portion. This latter alternative may be done temporarily by bringing the distal stump of the ileum through the parietes and tying in a Paul's tube, or permanently by implanting *both* ends of the divided ileum into the pelvic colon some inches apart.

The last case which I wish very briefly to call your attention to is one illustrating the conditions associated with long-continued chronic constipation. I say associated because I do not know whether these conditions are the cause or the effect of the constipation. The patient was an old woman, aged 78, whose body came to the Bristol Medical School for dissection. I found by inquiring from the workhouse where

she had lived that she had had much trouble with the bowels, the patient constantly begging for aperients, and when these were given she often suffered from troublesome diarrhoea. She had well-marked right-sided scoliosis and a dorsal dislocation of the right hip-joint which was the seat of a huge abscess. I imagine that this must have developed quite silently, because it had not been observed at all in the workhouse. In the abdomen the most conspicuous object was the colon, which was so distended as to measure 8 to 10 in. in circumference and was packed with hard fæces. I have here to-day the chief parts of the intestinal canal and stomach, in which the relations have been maintained as far as possible. The cæcum is 5 in. long and 2 in. in diameter, the appendix $3\frac{1}{2}$ in. long and adherent in the pelvis to the right Fallopian tube. The ascending colon and its hepatic flexure are bound into a complicated loop containing five distinct kinks something like the letter W, except that the various limbs of the loop do not lie in the same plane. The same membrane which binds together the limbs of this colic loop are continued over the gall-bladder, which is dragged down by this connexion and fixed to both the duodenum and colon. In the gall-bladder were some large gall-stones. The transverse colon is 14 in. long and about 3 in. in diameter. It presents a sharp kink about the middle, and this part lay in the cavity of the pelvis. From the splenic flexure onwards the gut presents nothing unusual. The stomach is remarkably distorted in a manner which presents an exaggeration of the normal curves and divisions. A marked subdivision exists between the cardiac and pyloric chambers, the former consisting of a globular cavity 5 in. by 4 in. when distended and the latter forming a canal which exactly resembles the small intestine except for its thicker walls. At the junction of these two parts of the stomach is a well-marked notch on the great curve, and below this the pyloric chamber formed a U loop, each limb of which is 3 in. long and only $\frac{1}{2}$ in. from its fellow. This remarkable kinking of the pyloric part of the stomach appears to be caused by the direct drag of the loaded colon pulling upon it by the gastro-colic omentum. The third part of the duodenum lay across the third lumbar vertebra, and there is a sharp bend between the first part, which runs nearly vertically up, and the second part, which runs nearly vertically down.

This specimen thus illustrates very well the conditions which have so often been described by Mr. Arbuthnot Lane as being associated with chronic constipation. They are:—

- (1) A matting together of the limbs of the colon.

(2) A marked pathological kinking of the colon at the hepatic flexure and at the middle of the transverse colon, and an exaggeration of the natural angle at the splenic flexure.

(3) A dragging down of the pylorus with kinking of the pyloric canal and of the duodenum.

(4) Biliary lithiasis.

(5) Adhesions of the appendix and of the uterine appendages.

(6) A slight degree of enteroptosis.

But the one special point which I wish to emphasize in this specimen is the nature of the so-called adhesions. These do not appear to have an inflammatory nature or origin, for the membrane which binds together the various loops of bowel is only loosely attached to them, and is in fact nothing more or less than the peritoneum whose relation to the gut has been altered by the enlargement of the latter. When the colon becomes dilated its peritoneal coat is partly stripped off, and in the case of the flexures it will run directly from the surface of one limb of the flexure to that of the other. And when the colon increases in length the peritoneum will run in the form of bands and folds across the intervals between the bulgings of the bowel. And the membrane running from the gall-bladder to the hepatic flexure is merely a common anatomical variation described in Poirier and Charpey's "anatomy" as the cysto-colic ligament. A similar peritoneal ligament may exist between the liver and the hepatic flexure—the hepato-colic ligament.

It seems to me to be of the utmost importance to recognize the fact that in idiopathic dilatation and constipation without ulceration of the colon inflammatory adhesions do not exist. And when these do occur it should lead to the inference that some other disease is present, *e.g.*, gastric ulcer or appendicitis, which may demand direct treatment, as the cause rather than the result of constipation.

The facts and conclusions to be derived from the foregoing may be summarized as follows:—

(1) That the colon absorbs about 10 per cent. to 20 per cent. of the water from the fæces.

(2) That this absorption takes place rapidly and is quite efficiently performed in the rectum and pelvic colon alone.

(3) That the absorption of foodstuffs in the colon is so slight as to be negligible.

(4) That therefore the greater part of the large intestine is functionally unnecessary.

(5) That as the contents of the colon consist of nearly one-third

part by weight of bacteria, the absorption of soluble bacterial products probably occurs in all cases of colic stagnation [14].

(6) In normal patients the whole of the colon can readily be irrigated by rectal injections, but this is doubtful when the colon is much dilated and kinked.

(7) The operation of ileosigmoidostomy or partial colic exclusion is in normal cases followed by no metabolic disturbance.

(8) Functional diseases of the colon associated with constipation are divisible into two main categories: (a) Idiopathic dilatation; (b) atony with secondary dilatation and kinking, but it is often difficult to distinguish between these in advanced cases.

(9) In cases of idiopathic dilatation of the colon, after an ileosigmoidostomy has partially excluded the large gut, this forms a blind pouch into which gas, fluid and fæces are forced by antiperistalsis. This necessitates the removal of this part of the colon.

(10) Cases of constipation from colic atony require great care in their discrimination between those suitable and those unsuitable for operation.

(11) An attempt should be made in all these cases to determine the place where stasis occurs by means of radiography after bismuth feeding [5].

(12) When the rectum and pelvic colon are the seats of stasis enemata ought to afford relief, and any operation will be of very doubtful utility.

(13) When the stasis occurs chiefly in the parts above the splenic flexure, then, if prolonged massage and medical treatment have proved unavailing, surgical treatment is necessary.

(14) When a patient is in an advanced condition of toxæmia it is probably making too great a demand on his strength to perform the excision and anastomosis at the first operation. The safer method would be to divide the ileum and perform an ileosigmoidostomy. The distal stump of ileum can either be brought to the surface and a Paul's tube tied in, or it may be implanted into the pelvic colon a few inches from the proximal part of the ileum.

(15) Walker Hall has observed that the quantity of intestinal ferments at the end of the small gut is much larger than in the rectal fæces.

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DISCUSSION.

The PRESIDENT (Mr. Warrington Haward), in thanking the authors for their paper, said it was not only of surgical, but also of physiological interest, and it would be very interesting to hear the experience of other members of the Section on exclusion of the colon and the effect on metabolism and nutrition of such operations as had been described.

Mr. MCADAM ECCLES said he thought there were two points of surgical and physiological interest in the contribution. One was that there might be an absorption of an almost equivalent quantity of water from the pelvic colon and the rectum as from the rest of the large intestine, showing that ileosigmoidostomy, or excision of the colon with ileosigmoidostomy, was not an operation which would put the patient in any physiological danger. The second point was that peculiar antiperistalsis occurred throughout the large intestine. He said "peculiar" because he did not think physiologists had shown that it occurred normally. It seemed to be always due to some abnormal excitement in the lower part of the large intestine. This led up to a very important point, to which Mr. Groves did not allude—*i.e.*, whether it was not better, in many of the cases where ileosigmoidostomy was performed for chronic constipation, to avoid dividing the ileum between the anastomosis and the cæcum, leaving the normal passage intact, taking care, however, that the opening between the ileum and the pelvic colon was sufficiently large to

enable three-fourths or more of the intestinal contents to pass direct into the pelvic colon. In some of his earlier cases he divided the ileum, and somewhat distressing symptoms ensued in a few. He thought Mr. Groves's explanation was probably right, that there was antiperistalsis in the colon and from the point of anastomosis and consequent accumulation of intestinal contents in the blind portion left behind. In his later cases he had not divided the ileum. If exclusion were done for malignant disease, the case was different. He thought that a certain amount of intestinal contents passing along the distal portion of the ileum and into the cæcum caused the requisite stimulation to produce peristalsis in the right direction. He desired Mr. Groves's opinion on the point.

Mr. LOCKHART MUMMERY said he could not agree with what Mr. McAdam Eccles said about antiperistalsis, as it was a quite normal function of the ascending and transverse portions of the colon. Leaving out experiments on animals, it had been observed in human beings by Hertz and others, and it had been seen to occur, by X-rays, at definite intervals after the ingestion of food. Last year he, Mr. Mummery, did a lot of experiments on cats, which were purged, and then given a large feed of meat. Then, under anæsthesia, they were put into a hot-water tank, the abdomen opened, and the intestines watched. After a certain time there was first contraction in the ileum, then antiperistaltic waves, starting in the centre of the transverse colon, and slowly sweeping back with a definite rhythm to the ileocæcal valve. Those changed to segmental waves. That went on for fifteen minutes and then stopped, being followed by segmentation for another five minutes. Then antiperistalsis would begin again. Those observations had been repeated by himself and others many times. Therefore he did not think the suggestion of Mr. Eccles as to not dividing the ileum would help in regard to ileosigmoidostomy. If one was to leave the colon, he thought a better method was to divide the ileum and also divide the colon above the anastomosis, establishing an opening on to the skin—*i.e.*, a left-sided colotomy with the blind colon. Washing out would then be possible, and the colon would have a reasonable chance of atrophying. Experimental work showed that the suggestion just made was incompatible with health of a permanent character, except in the few lucky cases where the colon completely atrophied. Experimentally, the colon had been partially obstructed above the anastomosis, and the effect of that was to simply postpone the time of recurrence of the mischief. The paper was a very instructive one, but it was necessary to be very careful before declaring that the colon was not a useful organ.

Mr. MCADAM ECCLES desired to explain that he was referring to antiperistalsis occurring from the pelvic colon upwards. He knew of the experiments referred to, but these, he believed, showed antiperistalsis only from transverse colon to cæcum. He was not sure there was evidence of antiperistalsis from the pelvic colon, except under abnormal conditions, such as liquid fæces flowing into the sigmoid.

Mr. GROVES, in reply, said the question of antiperistalsis had been sufficiently dealt with by Mr. Mummery. With regard to the advisability of treating such cases by lateral anastomosis, rather than by division of the ileum, the arguments against it were largely theoretical, but certainly Mr. Lane's first cases were treated in that way, and he gave it up as unsatisfactory. And many experiments had been done in which it was shown that lateral anastomosis, in the absence of definite stenosis, was generally followed by the intestinal stream going on in the old path, the best example of which was gastro-enterostomy done in the absence of pyloric stenosis, the gastric contents still going on in the old way. While one was at it, he thought the suggestion he made was a better one—namely, that the ileum should be divided, and the two ends implanted in the colon at some distance from one another. That seemed the most ideal procedure. With regard to ileosigmoidostomy not having been permanently successful, according to what he had read it had not been the cases which had had the colon excised which had suffered in that way, but the earlier cases which had been treated by simple ileosigmoidostomy. The patients he had referred to did all right, as far as the constipation went, for months, and then they began to lose flesh, to become anæmic and have pain. That was not due to the metabolic changes from short-circuiting, but to the fact that they had a great cesspool, in which there was no stream, and in which contents decomposed and toxic substances originated. With regard to excision of the colon, he did not think those observations showed that the whole colon need be excised, but he thought that if the symptoms in idiopathic dilatation of the colon in many of those cases were due to dilatation of the ascending colon and cæcum, it was clear there was no object in trying to remove the colon down to the rectum. The pelvic colon was 12 or 18 inches long, and if the ileum was anastomosed with the upper part of it, there would be sufficient colon left to carry on the metabolic economy. The point he had attacked was not whether the colon as a whole could be dispensed with, but whether, from a practical point of view, the greater part could be excluded or removed without any injurious metabolic effects. He thought the figures he had laid before the Section demonstrated that point.

The Repeated Strangulation of an Obturator Hernia necessitating its Radical Cure, with Remarks upon Obturator Herniæ in General.

By EDRED M. CORNER, M.C., and MARTIN HUGGINS, F.R.C.S.

WE are recording this case because the hernia was strangulated no fewer than three times, whilst the patient was never free from abdominal pain and discomfort in the intervals between these acute crises. Her clinical condition in consequence demanded that a radical cure should be devised and done. In addition the pathological condition of angulation of the bowel, the result of its repeated strangulation, pointed in the same direction. A radical cure was performed, and since that time she has been free from all discomfort.

Obturator herniæ are sufficiently infrequent for no individual surgeon to have an extensive experience of them. So that our knowledge of the condition has to result from the collected investigations of many observers. In order to keep this knowledge up to date the experiences recorded by the various authors have to be summed up from time to time. We have added to our case a summary of such knowledge as we could find and examine on the subject of obturator herniæ.

An obturator hernia is stated to be one of the rarities of surgery. This is hardly true at the present time (1908). Even in 1898 a writer says that over 200 examples were known and recorded. M. Jaboulay, in his text-book published in 1899, knew of 170. At the present time upwards of 250 cases are known; so that an obturator hernia has lost its claim to the title of a surgical rarity. The condition was first observed by Arnaud de Rentil in a case which was reported by Duverney in 1724. Reneaulene de la Garenne gave an account of a similar case in 1726, and in 1743 MM. Lacroissant and Garengéot added a third. Hence there is no doubt that the honour of recognizing obturator hernia belongs to the French surgeons. All herniæ from a clinical standpoint may be separated into two classes—the urgent and the non-urgent; the former compelling attention by their danger to life, the latter merely coming to notice through the expediency of having “something done.” Naturally, it is those cases which kill the patient or endanger his life which first receive attention. Thus most of our knowledge, particularly of recent years, has been gained from those surgeons who have operated

on cases of strangulated obturator herniæ. It is very infrequent for attention to be called for other reasons to an obturator hernia; one of the best modern examples, in which the size of the hernia attracted attention, is that recorded by Mason.¹ A third and a great channel for our knowledge of the subject is through the reports of observers who have noticed the hernia in the course of a post-mortem examination. The best modern example of this is undoubtedly that recorded by Lickley.² By means of these three channels of exploration it has been possible to add details of thirty-one more cases to those summarized by Berger and Jaboulay.

In order to gain some idea of the frequency with which obturator herniæ occur, the statistical reports of St. Thomas's and St. Bartholomew's Hospitals have been examined. From the twenty-one years, 1886-1906 inclusively, at St. Bartholomew's Hospital there were 970 cases of intestinal obstruction due to the strangulation of the bowel in the hernial regions—inguinal, femoral, umbilical, ventral, and obturator—of which only two were obturator, giving the proportion as one case in every 485 cases of strangulated hernia. During the seventeen years, 1890-1907 inclusively, there were admitted to St. Thomas's Hospital, 026 cases of strangulated herniæ, of which four were obturator, giving the proportion of 1 in 256 cases. If the statistics of both hospitals are put together the frequency becomes 1 in 332 cases. As St. Bartholomew's Hospital get on the average forty-six strangulated herniæ every year, they will get an obturator every seven years; and as at St. Thomas's they have sixty-four strangulated herniæ every year, they will have one obturator hernia every five years.

It is a form of hernia which is more common in women. Since the issue of Jaboulay's text-book in 1899 we have collected five cases in men and twenty-six in women, which combined with his figures give 144 women to twenty-three men. The approximate proportion is six to one, which is an extraordinary one, considering how much more frequent strangulated herniæ generally are in men as compared with women. From this great frequency one naturally suspects that the habit of child-bearing may influence the occurrence of the hernia. Unfortunately the records of the cases are not at all complete with regard to the sexual history of the women. And it is certain that in some cases no pregnancy had taken place. Yet the great majority of the female patients were married, and most of them, but not all, had borne children. Thus,

¹ *St. Bart.'s Hosp. Reports*, 1891, xxvii.

² *Glas. Med. Journ.*, 1903, lvii, p. 179.

though it would appear that the changes in the pelvis which result from pregnancy and parturition may dispose to the formation of an obturator hernia, there are other factors. For instance, one cannot but be struck with the description of the patient, so frequently repeated, being given as "wan, emaciated, and feeble"; and it is very possible that the larger obturator foramen in women becomes filled with a pad of subperitoneal fat which, when they become "wan, emaciated, and feeble," is absorbed—the removal of which support inviting the formation of an obturator hernia sac. In December, 1908, one of us operated on an emaciated man, aged 45, for intestinal obstruction. On each side an obturator hernia was present. Thus these herniæ are common at advanced ages, and are acquired. The oldest example recorded of recent years was aged 87, and the youngest, in the last ten years, aged 47. The cases of the last ten years may be arranged as follows:—

Aged between 80 and 90	...	8.5 per cent.
" 70 " 80	...	25 "
" 60 " 70	...	41 "
" 50 " 60	...	8.5 "
" 45 " 50	...	17 "

Thus it will be seen that obturator herniæ in subjects under 45 years of age are very rare. But they have been known to occur as early as the age of 12. As a general rule the men in whom it occurs are younger than the women.

Bilateral herniæ form 7 per cent. of the obturator herniæ; unilateral and left, 33 per cent.; unilateral and right, 60 per cent. The mortality is great for two reasons: in 70 per cent. of the cases only a part of the bowel is included in the strangulation of the hernia, the intestinal obstruction only being partial and incomplete, not demanding immediate relief; the majority of cases—I would suggest fully 80 per cent.—are undiagnosed. Under these circumstances temporizing measures are adopted and, the obstruction being incomplete, are persevered with, enemata yielding results and the sickness not being very troublesome. Thus operation is delayed until the strangulated bowel is gangrenous and it is too late to save the patient.

A recorded but unknown example of this has been published by Mr. F. C. Abbott.¹ It occurred in a woman, aged 70, who had suffered from abdominal pain and partial intestinal obstruction for no less than seven days before she was sent to the hospital. The abdomen was

¹ Special table of the Surgical Report of St. Thomas's Hospital, 1892, pp. 198-199.

explored at once and a piece of gangrenous bowel found strangulated so that its lumen was only partially obstructed, a partial enterocele, in the right obturator foramen. She never recovered from the operation, dying within twenty-four hours. In consequence the case has never been given a more prominent record and publicity, though its clinical lessons far outvalue those of many successful and prominently reported cases.

In every case of strangulated obturator hernia there should be local signs present, but they are often insignificant. A tender swelling below, deeper than, and rather internal to, the usual situation of a femoral hernia can be detected, especially when the two groins are compared. Frequently, but not in the majority of cases, the patients complain of pain down the obturator nerve. The real reason why this form of hernia is not diagnosed is that the method of rectal or vaginal examination is so frequently neglected. An obturator hernia, when strangulated, can be felt easily *per rectum*. In the last ten years the mortality has been about 33 per cent., but this is no criterion to go by, as the prognosis depends much more on the diagnosis being made early than on anything else; because nothing but operation can relieve the patient. Usually the diagnosis of intestinal obstruction is made, and the obturator hernia only discovered when the abdomen has been opened. Hence the result depends upon how much time is allowed to pass by before it is decided to operate.

The usual viscus found in a small sac of a strangulated obturator hernia is small bowel—ileum—which is partially engaged so that its lumen is only in part obstructed. No case has been known in which the large bowel was strangulated, though the sigmoid colon has been known to enter a left-sided hernia. There are several cases in which the ovary and Fallopian tube have been found in the hernia, such as those recorded by Lickley and Schopf. They differ in one great respect from such occurrences in inguinal herniæ. In the latter case the subjects are almost invariably young,¹ and in the obturator herniæ they are almost always old; for instance, Lickley's case was 87 years of age and Schopf's 68.

Regarded from the side, it may be said that inguinal herniæ are the most anterior, that obturator herniæ are the most posterior, and that femoral herniæ are intermediate. In accordance with this statement it is found that the anterior pelvic viscera, such as the bladder,

¹ *Brit. Med. Journ.*, 1908, i, pp. 17-19.

are most frequently in inguinal herniæ and least frequently in obturator herniæ. If the ovaries associated with their descent, such as are found in the inguinal herniæ of children, are disregarded, posterior pelvic viscera are most frequently found in obturator herniæ, less frequently found in femoral herniæ, and are least frequently found in inguinal herniæ.

Total number of cases examined	250
Average frequency for 2,000 cases of strangulated hernia	1 in 332
Proportion of obturator herniæ found in men and women	1 to 6, or 14·8 per cent. to 85·7 per cent.
Decade of greatest frequency of obturator herniæ	60 to 70 years
Oldest known patient	87 years
Youngest known patient	12 years
Right-sided obturator herniæ	60 per cent.
Left-sided obturator herniæ	33 "
Unilateral obturator herniæ	93 "
Bilateral obturator herniæ	7 "
Associated with incomplete intestinal obstruction (Richter's hernia)	70 "
Undiagnosed	about 80 "
Diagnosed	20 "
Mortality for operation on strangulated obturator herniæ for the last decade	33 "

REPORT OF CASE.

A married woman aged 62 was admitted under the care of Dr. C. R. Box to St. Thomas's Hospital in 1906. Up to five years ago she had been comparatively free from illness. She was the mother of six children and had had no miscarriages. Five years previously she was operated on by Mr. Carson at the Tottenham Hospital for acute intestinal obstruction. Mr. Carson kindly wrote to one of us stating that he did a laparotomy for intestinal obstruction and found a portion of the small bowel strangulated in the right obturator foramen. Since this operation she has had attacks of pain in the abdomen which were sometimes severe but never lasted any length of time. Three days before her admission to St. Thomas's Hospital, whilst at breakfast, she was seized with acute abdominal pain which gradually got worse, compelling her to go to bed later in the day. She was sick several times, particularly after trying to take food. The bowels were inactive, and no flatus was passed. In appearance she was a thin, pale woman with sunken eyes, who was easiest when lying on her back. The temperature was a little above normal; the pulse-rate 128, with a beat of small volume and tension. The vessels were thickened. The tongue was dry and covered with brownish fur.

On examination the abdomen moved evenly and fairly freely with respiration. It was moderately distended, particularly in the lower part. The scar of the former operation was to be seen below umbilicus. No tumour could be felt. The abdominal walls were not rigid, and there was no tenderness except in the right lumbar region (? referred from the obturator nerve, posterior division). A vaginal examination revealed a tender place on the right side of the pelvis. No inguinal, femoral or umbilical herniæ were found.

Operation was proceeded with at once, the incision being made through the old scar. The abdominal walls were very thin. On inserting two fingers towards the pelvis collapsed small bowel was found. This proved to be the lower part of the ileum. What proved to be its lower end was followed to the cæcum, whilst its upper end led to the right obturator foramen in which it was strangulated. The ensnared loop was released by a little manipulation, aided by flexing the thigh and pressure from without; no "herniotomy" being required. The hernia was then seen to be a partial enterocele or Richter's hernia. The patient's condition was now very bad, so that there was no thought of a radical cure for the hernia. The abdomen was filled with warm saline solution and the abdominal wall sutured in layers.

Her convalescence was uneventful. The bowels were well open on the second day after operation, and the wound healed by first intention. After leaving the hospital she was always having abdominal discomfort and pain, at the same time being much troubled with cystitis.

On December 19, 1907, she was readmitted to the hospital suffering from intestinal obstruction for the third time. Before leaving previously she had been warned to seek readmission at once if her symptoms of intestinal obstruction returned, and to waste no time in doing so. In consequence the symptoms were of less than twenty-four hours' duration. Abdominal section was immediately performed through the old scar. A part of the ileum was found to be ensnared in the right obturator foramen as a partial enterocele or Richter's hernia. It was easily disengaged without it being necessary to divide the neck of the sac to set free the bowel. It was then seen that where the bowel had been injured it had become adherent to itself, forming quite sharp angulation by the contact of the two limbs of the coil (fig. 1). It was decided not to resect the bowel to obviate this.

This operation was the third which had been performed on this patient for intestinal obstruction. In consequence, it was felt that something must be done to obviate any further occurrence of the accident.



FIG. 1.

To show the angulation of the bowel as the result of the repeated strangulation of the coil.

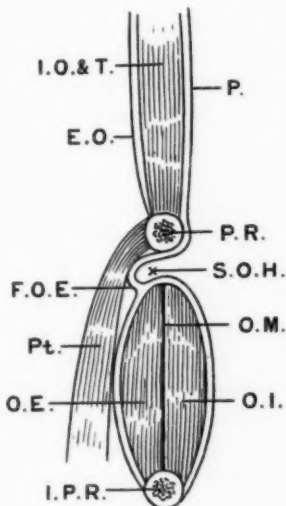


FIG. 2.

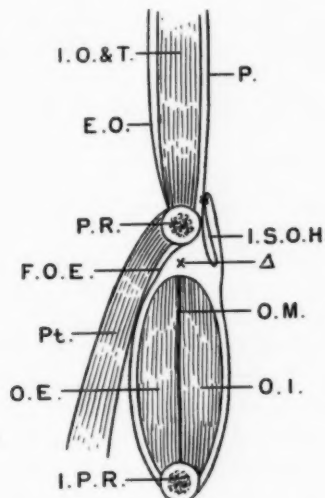


FIG. 3.

Fig. 2.—Diagrammatic section through the obturator foramen showing the hernia. E.O., external oblique muscle; I.O. & T., internal oblique and transversalis muscles; P., peritoneum; P.R., pubic ramus; S.O.H., sac of obturator hernia; F.O.E., fascia over obturator externus muscle; Pt., the pectineus muscle; O.M., the obturator membrane; O.E., the obturator externus muscle; O.I., the obturator internus; I.P.R., ischio-pubic ramus.

Fig. 3.—Similar section to that in fig. 2, showing the inversion of the sac. Lettering the same as in fig. 2 with the addition of I.S.O.H., inverted sac of obturator hernia; Δ, space previously occupied by sac.

An incision was made over the thigh, rather internal to the situation of an incision for femoral hernia, and parallel with it. The pectineus muscle was exposed, its fibres separated, and held apart with retractors. This exposed a layer of fascia—the obturator fascia—lying over the obturator externus muscle which concealed the sac. This fascia was strong and closed the outer opening of the obturator canal (fig. 2). It was incised, the sac being exposed and freed from its surrounding attachments, no trace of the pelvic fascia over the obturator internus being seen. It now became quite obvious that the sac could not be ligatured at its origin and a radical cure performed from the outer end of the obturator canal with any hope of success in preventing the recurrence of the hernia. The obturator foramen is converted into a canal by the obturator muscles, and it is no use at all to close the outer end of the canal to prevent anything entering the inner end, any more than it would be to close the back door of a house to prevent anyone coming in through the front door (fig. 2). So having freed the hernial sac through this crural incision, it became obvious that the radical cure must be completed through the abdominal incision. The unopened hernial sac was inverted, in a manner similar to that done in Kocher's radical cure for inguinal hernia, and maintained in the new position by a stitch through the parieties (fig. 3). The incision in the relaxed obturator fascia and the rest of the crural wound were closed. The abdominal incision was likewise sutured. Both wounds healed by first intention, and the patient was discharged from hospital sixteen days after operation. Since then she has been seen from time to time and has improved immensely in her general health, having suffered from no abdominal pains or discomfort. In fact, as she puts it, she has been in better health and bodily comfort than she has been for some years. This is a very interesting contrast to her general condition after her first and second operations, showing the advisability of following the relief of the strangulation by the inversion of the hernial sac, and also that the consequences of a considerable degree of angulation of the bowel (fig. 1) by adhesions can be neglected.

To sum up, there are, we think, two main points in the acquisition of an obturator hernia. The first and most important is that the patient, after being previously, and perhaps only comparatively, fat, has become thinner. With the general absorption of the fat throughout the body, that within the obturator canal disappears and is replaced by the bulging of the peritoneum over it to form the hernial sac. Thus a very important part of the post-operative treatment of these cases is to fatten them.

This cannot always be done if a radical cure for the hernia has not been performed, on account of the abdominal discomfort probably caused by the temporary lodgments of the bowel in the hernia. In the case here reported the patient has gained flesh since the last operation, and it is hoped that in the general acquisition of fat some of it has been deposited in her right obturator canal between the obturator fascia and the inverted hernial sac so as to give stability and permanence to the radical cure. The second most important factor in the production of an obturator hernia is the enlargement of the obturator canal by the changes which take place in the pelvis in consequence of pregnancy. Thus, women are six times more frequently subjects of this hernia than are men, and the great majority of them are multipara, the *bien-être* of the pregnancy with its fattening followed by the strain of lactation and its loss of flesh powerfully aiding these changes.

As further example of the advisability of following the operation for relief of the strangulation by one for radical cure a case may be quoted as recorded by Mr. C. A. R. Nitch.¹ A man, aged 67, was operated on for incomplete intestinal obstruction of one week's duration. A left-sided strangulated obturator hernia was relieved; a partial enterocele of ileum. Recovery followed. No radical cure was done. Six weeks after leaving hospital the obstruction returned, but as the patient was taken elsewhere it is not known if he recovered or died.

Reference must also be made to the subject of reduction of herniæ *en masse* in obturator herniæ.² Dr. Tonking, of Camborne, has recorded the spontaneous reduction of a right-sided strangulated obturator hernia; the sac containing a coil of small bowel which had apparently been reduced *en masse* by the vigorous peristalsis of the obstructed bowel. It was found when exploring the abdomen for intestinal obstruction.³

From the collected experiences of observers it would appear that the relief of a strangulated obturator hernia should always be followed by a radical cure when feasible, either at the same or a later operation. The simplest operation for radical cure consists of freeing the sac through an incision in the thigh, invaginating it, and maintaining it in its new position by a stitch through the abdominal walls by means of a coeliotomy wound through the right rectus abdominal muscle. Two incisions, an abdominal and a crural, are required to perform a satisfactory radical cure such as cannot be done through one or either incision.

¹ *St. Thomas's Hosp. Reports*, 1904, pp. 213-214.

² Corner and Howitt, *Annals of Surgery*, April, 1908, pp. 573-587; and *St. Thomas's Hospital Reports*, 1906, pp. 439-539.

³ *Lancet*, 1904, ii, 917-918.

Obturator herniæ shed a sidelight on the popular and comfortable theory of all herniæ being caused by an early and congenital formation of the sac. They are rarely ever congenital, and in the vast majority of instances are acquired herniæ; loss of fat and repeated pregnancies being the two chief factors in their production.

DISCUSSION.

The PRESIDENT (Mr. Warrington Haward) said he was sure the Section wished to accord its best thanks to the authors for their interesting paper. Anything which added to the surgeon's capacity for diagnosing obturator herniæ must be of great use. He took it that the high mortality in connexion with the condition of strangulation was due to its non-recognition and to surgeons not seeing such cases until long after strangulation had occurred. The difficulty in diagnosis was enhanced by the fact that the classical symptoms of strangulation were sometimes absent in obturator herniæ. For instance, there was the absence of constipation, which he supposed was often due to only a small part of the calibre of the bowel being included in the sac. And in very stout people it was difficult to detect a tumour in Scarpa's triangle. It was no doubt very useful to be reminded, as we had been by the authors, of the additional aids to diagnosis obtained by examination *per vaginam* and *per rectum*.

Mr. ARTHUR BARKER said he only rose to ask a question of the authors of that exceedingly interesting paper—namely, whether they thought, from what they saw in the case, that it would have been possible to invert the sac without cutting into the thigh. He asked that because he recalled a case many years ago in which he operated upon a patient for obscure intestinal obstruction, and found that a very small femoral hernia had been reduced *en masse*. He found it in the usual situation, and withdrew it after division of the neck of the sac. It was then easy to put forceps down into the sac, seize the base of it, and invert it, tie a thread round the neck, and produce a radical cure of the hernia. If that could be done in obturator hernia, it would reduce the time spent in the operation, and hence also, of necessity, the risks. Could the authors have done that without doing the operation through the thigh, and cutting through the pectineus muscle, which was always a difficulty, and took time?

Mr. MCADAM ECCLES said congratulation was due to the authors on two points: first, on their able paper, and secondly, on the brilliant surgery which saved the patient's life no less than three times. The authors had said that at the usual rate a case of obturator hernia should occur at St. Bartholomew's every seven years. Curiously enough, this period arrived last September, and he had a case, which was duly recorded in the Journal of that hospital. The

patient was a married woman, aged 59, who was admitted because of pain in the abdomen. She had been subject to constipation all her life, and during the last three years had had occasional attacks of abdominal pain, accompanied by constipation and vomiting, the attacks lasting several days. Fourteen days before admission she experienced sudden, acute pain in the centre of the abdomen which compelled her to cease work. She only vomited once. On the following day the abdomen was much distended, she was sick several times, but the pain was rather better. During the following five days the distension still persisted, and she occasionally vomited after food had been taken by the mouth. The bowels were opened daily. Vomiting had been incessant until the day before admission, when it suddenly ceased, and the pain was localized to the umbilicus. The patient said she brought up the vomited matter without effort. Five days before entering the hospital she had been fed by nutrient enemata, and had taken nothing by the mouth. She was pale and greatly emaciated, and showed all the signs of collapse, with a temperature of 97° F.; she was very ill indeed. The abdomen was very poorly covered, and distended coils of bowel were clearly seen, with peristalsis in them. Rectal and vaginal examinations were made, but without anything definite being found. It was not always easy to diagnose strangulated obturator hernia by even these examinations. He operated, as Mr. Corner did in his case, and found some collapsed intestine in the pelvis, and on tracing it he came to the right obturator region. Unfortunately, with only a gentle pull during examination, the bowel came away, there being an area of gangrene about the size of a threepenny-piece. This was dealt with by excision, but the patient died shortly after the operation. He thought Mr. Corner would agree that in the majority of these cases the hernia was an extremely small one, and he doubted whether, notwithstanding the statement in the text-books, it could be diagnosed by a tumour in the thigh. Without doubt, the records of all these patients showed that when a patient had obscure abdominal symptoms, even if there were not complete obstruction, the sooner laparotomy was performed the better.

Mr. HEY GROVES desired to refer to the relation of the neck of the sac to the obturator foramen. He asked whether the authors considered that the obturator hernia always came through the same aperture that the obturator vessels and nerves did, or whether it bulged its way through a weak place in the membrane. He did not gather that that was the meaning of the authors, as they several times referred to the obturator fascia as if it were one of the coverings of the sac. If the hernia left by the same opening as the vessels and nerve, it followed that the hernia ran above the obturator fascia, which formed the line of fascia below the canal. That raised the point as to whether it was the tense band there which was the strangulating agent.

The PRESIDENT said that probably one reason for accepting the statement that the bowel did pass through the obturator canal proper was the frequent association of pain along the course of the obturator nerve, as was pointed out long ago. Probably that had been sometimes one of the most useful signs for diagnosis.

Mr. CORNER, replying on the discussion, said the patient in question chanced to turn up at the hospital a few days since, and he was able to satisfy himself that she had enormously improved in health, and was getting quite fat. The paper stated that a very important factor in the causation of obturator herniæ was the loss of body fat, and that was supported by the case narrated by Mr. McAdam Eccles. He (Mr. Corner) recently had the opportunity of making an observation on a very thin person—viz., a man, aged 45, who attended St. Thomas's Hospital on account of intestinal obstructions. He was very emaciated indeed, and having in mind the present paper, he had the curiosity, before closing the abdomen, to examine his obturator foramina; and he found that both of them were sufficiently large to admit the tip of his finger. Therefore, that man, most probably as the result of his emaciation, had a potential bilateral obturator hernia. There had been a tendency in recent years to regard most herniæ as congenital—due to developmental defect—not as acquired. But he could say that it was very unusual for an obturator hernia to be congenital. In fact, an obturator hernia is an acquired condition. With regard to Mr. Barker's question, concerning possible inversion of the sac through the abdominal incision only, that was tried first, before the crural incision was made, but it could not be done. In Mr. Barker's case he understood it had already been partially reduced *en masse* (Mr. BARKER: Yes), which was a great help. In answer to Mr. McAdam Eccles, the amount of bowel engaged was very small; and in about 70 per cent. of the cases only a part of the bowel was included in the hernia, the result being a partial enterocoele. The presence of a tumour in the thigh in such cases was very rarely recorded, so that it might be taken to have been seldom present. It was explicable by the small amount of the bowel implicated. The diagnosis in most of the cases was not made until a laparotomy was done, and in former days post mortem. In answer to Mr. Groves, in the case dealt with in the paper, and in that he had since mentioned (of intestinal obstruction), the hernia was through the obturator canal; and the recorded cases showed that the hernia in all cases had been through that canal, not through a weak part of the obturator membrane. On one point knowledge was as yet imperfect—namely, the relation of the obturator hernial sac to the obturator vessels and nerve. That was important, because there was always the possibility of having to divide some constricting band so as to free the ensnared bowel, and it was important to know in which direction to do that. In the majority of cases it seemed clear that the hernial sac lay above the obturator vessels. In answer to the President, concerning pain along the obturator nerve, he had been disappointed to find how conspicuously absent that seemed to have been; the small size of hernia explaining the absence of serious pressure on the nerve, as it explained the absence of a tumour clinically.

Surgical Section.

February 9, 1909.

Mr. J. WARRINGTON HAWARD, President of the Section, in the Chair.

The Lymphatics of the Colon.

By J. KAY JAMIESON, C.M., and J. F. DOBSON, M.S.¹

THE following description of the lymphatics of the colon is the result of an examination of twenty-three specimens injected with Prussian blue after the method of Gerota. We have already published an account of the lymphatic anatomy of the appendix, the cæcum, and the lower part of the ascending colon [7]. The first part of the rectum is included in this paper, but we have not yet been able to inject a sufficient number of specimens of the rest of this organ to enable us to describe its lymphatics. We have modified the technique of the injection by making use of a simple pressure apparatus which is more convenient to manipulate and which permits better control of the pressure than the hand syringe with which our previous injections were made. Though numerous coloured materials have been tried none have proved so satisfactory as Gerota's suspension of Prussian blue in turpentine and ether.

We propose to deal first with the arrangement of the blood-vessels to facilitate the description of the lymphatics, and because in dealing with the operative surgery of growths in the colon we shall frequently be under the necessity of discussing the details of the arterial distribution.

The superior mesenteric artery supplies three branches to the large intestine—the ileocolic, the right colic, and the middle colic. The relation of the ileocolic artery to the lymphatic vessels has been considered in a former paper [7].

¹ From the Anatomical Department of the University of Leeds.

The right colic artery is a direct branch of the superior mesenteric in less than 50 per cent. of subjects. In about 30 per cent. it is a branch of the ileocolic, and in other cases it is represented by a branch of the middle colic and an enlarged branch of the ileocolic.

The middle colic artery springs from the superior mesenteric artery in front of the head of the pancreas at, or a little distance below, the lower border of the body of the gland. It first passes downwards and outwards across the superior mesenteric vein, sometimes in front, sometimes behind this vessel, and then enters the transverse mesocolon, between the leaves of which it runs downwards, forwards, and to the right towards the hepatic flexure. About midway between the root of the mesocolon and the colon it divides into two branches, right and left, which diverge from each other, and each of which divides again close to the colon. There are, therefore, four secondary branches, which form three arcades—one with the ileocolic or right colic under the hepatic flexure, a second short arcade under the right end of the transverse colon, and one with the left colic artery. The last arcade is the longest and most important to be found along the intestinal canal; it is normally single, lying one or two finger-breadths from the gut, along the left two-thirds of the transverse colon, and enclosing in its concavity a large, conspicuous area of mesocolon, in which there is no vessel of any size. In some cases the middle colic merely divides near the hepatic flexure into two branches, forming two arcades, with the ileocolic or right colic on the one hand and the left colic on the other; in such case there is no middle arcade, and the great left arcade is longer than usual. The junction of the middle colic and the left colic arteries may generally be detected as being the point where the arching vessel is least in diameter, and this point is usually near the junction of the middle and left thirds of the transverse colon. A variable part of the ascending colon, the hepatic flexure, and the right and middle thirds of the transverse colon must be considered as the normal area of distribution of the middle colic artery. Secondary arcades between the vessels arising from the primary arches are infrequent in occurrence and inconstant in position—more frequent, however, under the hepatic flexure than along the ascending or transverse colon. Occasionally accessory middle colic arteries are found, and in a few cases there is a second arcade between the middle and left colic arteries along the curved upper portion of the inferior mesenteric vein.

The inferior mesenteric artery arises from the abdominal aorta under cover of the duodenum, from $1\frac{1}{2}$ in. to 2 in. above the bifurcation. It

descends, inclining to the left, in front of the aorta, the left psoas muscle, and the left common iliac artery, to enter the mesorectum; when it reaches the common iliac artery it becomes known as the superior hæmorrhoidal artery; it is accompanied in the lower part by the inferior mesenteric vein, which leaves it on the psoas to pursue an independent course. The left spermatic vessels lie about a finger's

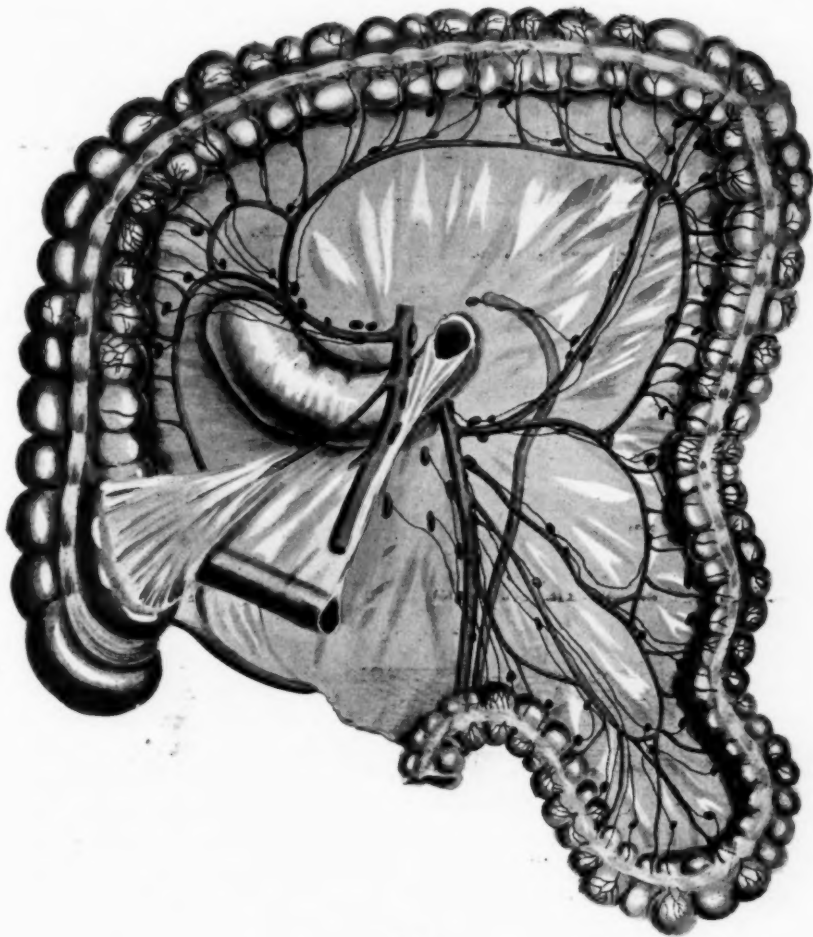


FIG. 1.

The lymphatics of the colon.

breadth to its outer side, and the left ureter descends on its outer side, gradually approaching it until, just above the common iliac artery, it lies alongside of it or may even bend in behind it; it is covered only by the peritoneum and the subperitoneal tissue, and it is surrounded by a sympathetic plexus; its branches are all given off from its outer side, and consist of the left colic artery and the sigmoid arteries.

The left colic artery is a large branch arising from the main trunk just as it issues from behind the duodenum. It runs outwards about an inch towards the lower pole of the kidney, and then meeting the inferior mesenteric vein, it turns sharply upwards and outwards across the kidney towards the splenic flexure; opposite the hilum of the kidney, the vein turns inwards across the artery, and thereafter the artery is accompanied by the left colic vein. At the lower border of the pancreas it turns forwards into the left extremity of the transverse mesocolon, and ends by dividing into two branches at or to the right of the splenic flexure; the right branch forms with the middle colic the great arcade along the transverse colon; the left bends downwards to form either a long arcade with the highest sigmoid artery or short arcades with its own branches. The branches of the artery vary in number from one to four. The highest sigmoid artery frequently arises from the left colic, and other sigmoid arteries may have their origin from it either separately or by a common stem. In most cases one or more branches run across the kidney and form a line of arcades along the inner side of the descending colon, with each other, with the left terminal branch and with the highest sigmoid artery.

The sigmoid arteries, one to four in number, arise from the inferior mesenteric or the left colic, or both. The highest branch, arising from the left colic or the inferior mesenteric stem, runs outwards between the lower pole of the kidney and the iliac crest, and supplies the lower end of the descending colon and that part of the gut which is often designated the iliac colon. The remaining sigmoid arteries, arising irregularly from the stem of the inferior mesenteric, are always to be found descending in front of the psoas to enter the mesosigmoid. The lowest sigmoid may be found close to the outer side of the common iliac artery. All these branches form simple arcades with each other in the mesosigmoid, but it must be noted that the lowest sigmoid and the superior hæmorrhoidal arteries do not anastomose in the same way as the individual sigmoid arteries; they stand in relation to each other as the diverging limbs of two

arcades, and the essential part of the communication between the lowest sigmoid artery and the superior hæmorrhoidal artery is at the point of origin of the former. Reference to the diagram below (fig. 2) will show that if the main arterial trunk is ligatured below the origin of this artery, blood could not reach the lower part of the gut through the system of arcades, whereas if the artery is ligatured above the point of origin the blood-supply is still effective. [Manasse 11.]

Along the great intestine we have never met with an instance of failure in the formation of anastomotic arches between neighbouring colic vessels, but the inosculations are single, secondary arches being found constantly only at the flexures.

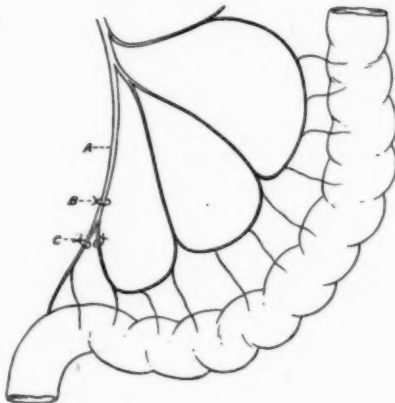


FIG. 2.

A, inferior mesenteric artery; B, ligatures without destroying anastomosis;
C, ligature breaking the anastomosis.

The superior hæmorrhoidal vein, as it crosses the common iliac artery, becomes the inferior mesenteric vein. It ascends along the outer side of the artery for about 2 in., crossing the sigmoid arteries. It then leaves the artery, proceeding upwards to join the left colic artery. It accompanies this artery for an inch or two, and then turns sharply upwards over or under the artery and forms a well-marked and conspicuous arch below the pancreas, above the duodeno-jejunal flexure, which ends by disappearing under the pancreas to join the splenic or superior mesenteric vein. It receives the tributaries corresponding to the branches of the artery.

The lymphatic glands which receive lymph from the large intestine are scattered along the course of the blood-vessels and are variable in size and number. Though compared with the glands of the mesentery proper they may seem few, yet taking the length of the intestine, which they drain, into consideration, they may be said to be proportionally as numerous. As they are not collected between the leaves of a mesentery, they must be described in chains corresponding to the vessels along which they lie; the ileocolic chain, the middle colic chain, the left colic chain, and the inferior mesenteric chain.

As the right colic artery is inconstant, and as, when present, few glands are found along it, there is no need to interpolate a right colic chain between the ileocolic and the middle colic. In each chain we find that the glands tend to form groups at certain points, and though, owing to the presence of scattered glands between these groups, any subdivision of a chain is arbitrary, it is convenient for purposes of description and reference. In each chain we shall distinguish epicolic, paracolic, intermediate and main groups.

The epicolic glands are those which lie on the intestinal wall under the peritoneum and in the appendices epiploicæ. Of small size they are very numerous in young subjects, but decrease in number with age. Though found on any part of the large intestine, they are especially numerous on the sigmoid flexure. They receive some of the vessels which issue through the muscular coat, and send their efferents to the paracolic and intermediate groups.

The paracolic glands lie along the inner margin of the gut, from the ileocolic angle to the rectum, mainly between the intestine and the arterial arcades and on the arcades. Though more numerous in the young, they are found at any age, and the average size may be given as that of a small pea.

The intermediate groups lie around the stem of the middle colic artery and the left colic and sigmoid branches of the inferior mesenteric. They tend to group themselves about the arteries midway between their origins and the gut, though in any chain between this group and the paracolic group, on the one hand, and the main group, on the other, there are connecting glands. On the middle colic artery the group is to be looked for midway between the root of the mesocolon in front of the pancreas and the hepatic flexure, on the primary bifurcation of the artery, but not infrequently it is nearer the origin of the artery and continuous with the main group. On the left colic artery the chief mass of the intermediate glands is seen just above

the point of crossing of the inferior mesenteric vein in front of the hilum of the kidney.

On the upper sigmoid arteries the intermediate glands lie in the retroperitoneal tissue, between the crest of the ileum and the lower pole of the kidney; on the lower arteries they lie in the base of the mesosigmoid. They vary considerably in their relation to the intestine; they may be situated close to the colon, and are only to be distinguished from the paracolic glands by the fact that they lie on the sigmoid arteries before these vessels divide; on the other hand, some members of the group may be placed so near the origin of the sigmoid arteries as almost to justify their inclusion in the main group. We have applied the term intermediate to all such glands on the sigmoid arterial stems which could be removed without exposing the inferior mesenteric artery.

The intermediate groups receive most of the efferents of the paracolic glands and some vessels from the gut which have slipped past these glands. The frequency of the occurrence of direct vessels to intermediate glands varies in different regions. From the hepatic flexure, the upper end of the ascending colon, and the right extremity of the transverse colon, they were found in every well-injected case (eleven specimens); from the splenic flexure, in only four cases out of fourteen specimens; from the transverse colon in its central portion not at all, and from the descending colon in five out of fifteen specimens. The sigmoid flexure resembles the hepatic flexure, and direct vessels to intermediate glands were constant. The efferent vessels of the intermediate glands pass to the main groups.

Main Groups.—The main middle colic group lies on the middle colic artery as it enters the transverse mesocolon. It forms a well-marked, readily recognizable mass, occasionally increased in size by confluence with the intermediate group. Usually it is clearly marked off from the great glands of the superior mesenteric chain, but sometimes it merges with them. The main group receives the efferents of the intermediate glands, some efferents of the paracolic glands, and frequently (five times in fourteen specimens) vessels from the intestine itself.

The main left colic group is divided into two sets. One lies mainly around the horizontal part of the artery, near its origin, and is continuous with the inferior mesenteric main group above the origin of the left colic artery. It receives some of the efferents of the left colic intermediate group, as well as efferents of the paracolic and intermediate glands on such branches of the left colic artery to the

descending colon as may be present; we have never seen a single instance of a direct vessel from the gut reaching this main group. The second set of glands lies on the terminal portion of the inferior mesenteric vein, in front of the head and at and under the lower border of the body of the pancreas, to the left of the superior mesenteric root-glands. This somewhat straggling group receives some of the efferents of the left colic intermediate glands, but never a direct vessel or even a paracolic efferent. Its members are small, and their efferent vessels communicate with the superior mesenteric glands and with the coeliac and lumbar glands.

The inferior mesenteric main group lies around the stem of the inferior mesenteric artery, as far up as the left colic artery, where, joining the main left colic group, it becomes continuous with the lumbar glands. Its members are neither numerous nor large, and are found not only on but under the artery. Its relations to the intestine are complicated, because it is only the upper part of the group which is comparable to any of the previously named main groups—*i.e.*, receiving paracolic and intermediate efferents and occasionally direct vessels. Owing to the way in which the inferior mesenteric artery and the intestine approach each other to meet at the pelvic brim, the lower glands of the main group receive efferents of the paracolic glands along the lowest part of the sigmoid flexure, and very frequently direct vessels, and stand in the same relation to the gut as the intermediate glands in other regions, while the lowest part of the group is almost in contact with the extremity of the sigmoid flexure and the first part of the rectum, and receives the majority of the direct vessels from the gut, thus corresponding to the paracolic groups elsewhere (fig. 3). Though some of the efferent vessels of the glands of the lower part of the chain run along the artery into the upper glands just below the origin of the left colic artery, it is important to note that the inferior mesenteric group, from its very lowest part upwards, sends a stream of efferent into the lumbar glands, not only to those on the left side of the aorta, but to those in front of it and between it and the vena cava; so that the inferior mesenteric group is not continuous with the lumbar group at its upper end only, but along its whole length, and is better regarded as a lateral extension of the lumbar group. Having in mind the fact that the lower glands of the chain are intermediate in character in their relation to the gut, and the lowest glands are paracolic, it is obvious that as we descend the lumbar glands become more and more nearly related to the gut.

The inferior mesenteric main group receives the efferent vessels of the intermediate group on the sigmoid arteries, some efferents of the paracolic glands and direct vessels from the sigmoid flexure. (Seven instances in eighteen specimens.)

The lymphatic vessels arise from plexuses in the submucous and subperitoneal coats, which have the same disposition as in other parts of the alimentary canal. The deeper vessels escape chiefly along the entering blood-vessels, and those from the outer side of the gut curve behind the gut to join those from the front. Many are interrupted almost immediately in the epicolic glands in the appendices epiploicæ or on the sacculi. The efferents of these glands, with other unin-

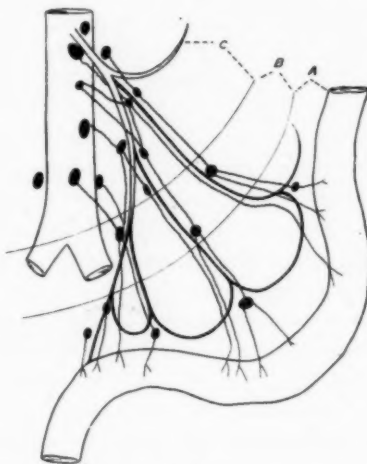


FIG. 3.

Showing how the lowest and middle glands of the inferior mesenteric chain are homologous with the paracolic and intermediate groups on the sigmoid arteries. A, paracolic group; B, intermediate group; C, main group.

interrupted vessels, pass inwards and meet the paracolic glands which intercept the great majority, but some vessels slip between the paracolic glands and reach the intermediate glands in company with the epicolic and paracolic efferents; in some cases these vessels run directly to the main group glands. A survey of all the specimens brings out the fact that from the diagonally opposite flexures, the hepatic and the sigmoid, these direct vessels to the main groups are more common than from the intestine between these flexures; from the splenic

flexure vessels never get beyond and rarely so far as the intermediate group. The lymphatics run with the blood-vessels, and only exceptionally in the avascular intervals; in the case of the transverse colon the vessels run in one direction or the other for a considerable distance, threading their way among the numerous paracolic glands. They are interrupted by these glands, so that it is not surprising to find that from the left third of the transverse colon few vessels reach even the intermediate group on the left colic artery, or that from the middle third no vessels are seen to reach the intermediate group on the middle colic artery. In the left half or two-thirds of the transverse colon there is an additional outlet for the lymph. In well-injected specimens we find that vessels arise from the anterior surface of the colon, run into the gastrocolic omentum, and then turn to the left between the ascending layers of this fold to reach, finally, the glands at the hilum of the spleen. The vessels from the middle of the transverse colon are necessarily very long, and there may be small interrupting nodules on them. We have also noted in two cases vessels ascending from the back of the upper end of the descending colon (lower limb of the splenic flexure) to the splenic glands. In Poirier's text-book [14] it is mentioned that the vessels of the transverse colon come into relation with those of the stomach in the omentum, but the vessels we have seen do not run with the epiploic branches of the gastro-epiploic arteries to join the vessels proceeding from the stomach; they run transversely to the left parallel with, but at a much lower level than, the left gastro-epiploic lymph-vessels, and cannot be confused with them.

Some of our specimens show evidence of communication between the lymphatic and venous systems in the abdomen. While injecting the region of the splenic flexure we noted that, after the chain of glands extending along the left colic artery and the arched portion of the inferior mesenteric vein had filled, the fluid appeared in the terminal end of the inferior mesenteric vein and in the superior mesenteric and splenic veins. We were unable to find a lymphatic vessel passing from one of the glands into a vein; probably the necessary manipulation emptied the vessel. The fluid did not enter the veins by puncture of their radicles at the seat of injection, because it first appeared in the distal ends of the great trunks, and no matter how distended by injection a gland may be we have never been able to force the fluid into the veins proceeding from the gland. We conclude, therefore, that there must be communications between the

glands about the head of the pancreas and the great venous trunks. Leaf [9] has shown that there may be communications between lymph-vessels and veins, and embryologically it has been demonstrated that the lymph-system is connected with the venous system at other places than the root of the neck.

When we come to consider the incidence of disease of the lymphatic glands of the colon we may confine our attention, for all practical purposes, to their affection in cases of new growths. A search through the innumerable monographs and articles on the subject of carcinoma of the large intestine shows how little we know of the subject of invasion of the lymphatic glands in this disease. This no doubt is in part due to the fact that the disposition of the different groups of glands has been imperfectly understood, and in part to the excessive amount of labour involved in accurately determining the presence or absence of malignant disease in a large number of glands. Here, as elsewhere in the body, carcinoma gives rise to secondary growths in the associated lymph-glands, but it is generally accepted that this invasion is not invariable, and when it does make its appearance it is at a late stage in the progress of the disease. Many writers have emphasized this view and have pointed to cases in which an advanced primary growth has been found without any sign of glandular metastasis (*vide* Treves [16], de Bovis [5]).

In other cases the lymphatic glands have been found enlarged and hard, but on microscopical examination have presented no signs of malignant infiltration. This has occurred in the experience of numerous observers, Bilton Pollard [15], Lennander [10], Hartmann [6], H. S. Clogg [3], and others. There can be little doubt that this hypertrophy of the associated lymph-glands is due to the fact that absorption takes place from the ulcerated surface of the growth and from the dilated, inflamed, and frequently ulcerated gut above the point of obstruction. As de Bovis remarks, the glandular hypertrophy does not occur only in the zone adjacent to the intestinal neoplasm.

It is a difficult matter to obtain reliable evidence of the frequency of lymphatic invasion in the early stages of the disease; in advanced cases seen in the post-mortem room metastasis in the glands is sufficiently obvious, as also in the viscera and the peritoneum. A certain amount of information is to be obtained by studying the late results of excision of the growth without any attempt to excise the "lymphatic area." De Bovis has made an exhaustive study of the cases reported in the literature and has tabulated the results. Combining his tables we find that of 102 cases which recovered from the operation of colectomy,

twenty-nine—i.e., 28·4 per cent.—either died from recurrence or were thought at the time the case was published to be suffering from recurrence; and this takes no account of the possibility of recurrence taking place in those cases reported shortly after operation, or of cases in which the late result is not recorded. Cavaillon and Perrin [2] quote Mickulicz as having ten recurrences in twenty cases following after colectomy. If we grant that in some cases the recurrence is due to imperfect removal of the primary growth, and that in some visceral deposits were not detected at the time of operation, it must be admitted that the majority were in all probability due to the presence of metastases in lymphatic glands which were not removed.

Mr. H. S. Clogg [3] has drawn attention to the frequency with which local and visceral recurrences occur after colectomy. In his view a local recurrence is usually due to the persistence of growth in the glands, and late recurrences in the liver are to be explained by cancer-cells lying dormant in the glands for some time and then for some reason being carried to the liver. Mr. Clogg [4] has recently published the results of the microscopical examination of the lymphatic glands in forty-five cases of malignant disease of the cæcum and colon. In twenty-eight cases cancerous deposit was found in the glands, and it may be that some of the negative cases might have shown evidence of disease had serial sections of all the glands been examined.

It is evident that in some cases of carcinoma of the colon in the early stages the lymphatic glands will be found free from disease. If the growth be removed at this stage, even though a purely local excision be performed, there is a considerable prospect of freedom from recurrence. It must, however, be allowed that the glands will become affected at some stage of the disease, and in a certain proportion at an early stage. Further, there are no means short of microscopical examination of determining when a gland is or is not affected with malignant disease. This is shown by the experience of Mr. Bilton Pollard where definitely enlarged glands were found to be free from growth, and by the case of Mr. W. Thompson, which we describe below, where apparently healthy glands contained growth. It is impossible for the surgeon to determine at the time of operation whether a growth in the colon has given rise to secondary gland infection or not. To minimize as far as possible the likelihood of recurrence it is desirable, then, to remove with the primary growth those glands which are likely to have become affected. The primary glands—those receiving direct vessels from the gut—will be the first to become affected by cancerous emboli carried to them along the

lymphatic vessels from the primary growth, and any one primary gland is just as likely to become affected as another, whatever may be their comparative position with regard to the colon.

We have had the opportunity of examining serial sections of the lymphatic glands in a case of carcinoma of the sigmoid flexure, the result of which is so striking, and so fully bears out the necessity of removing the lymphatic glands, that we append the details. The material was procured from a case of intestinal obstruction under the care of Mr. W. Thompson. The growth was a small one encircling the gut in the upper part of the sigmoid flexure. To the inner side of the growth lay a small gland of the size of a pea, soft and apparently healthy; on the uppermost sigmoid artery, just below the lower pole of the kidney, there lay another gland, of the intermediate group, hard, yellow, and adherent to the peritoneum; a third gland lay on the outer side of the inferior mesenteric artery just below the origin of the left colic artery and was soft and normal in appearance. No other glands were found in the vicinity of the tumour or along the vessel supplying the part involved. Serial sections of the glands and of the tissues of the mesocolon between the growth and the glands were cut. The primary tumour was a typical adenocarcinoma. The paracolic gland was riddled with minute masses of growth; the intermediate gland was converted into a mass of tumour tissue, the ordinary gland tissue being only seen in places; the main group gland, which was so placed in the microtome that the upper end was cut first, showed no trace of growth until the extreme lower end was reached, where in the entering end of a vessel a plug of cancer-cells was found. As the large lymphatic vessels reach the glands they are seen to break up into branches before entering the peripheral lymph sinus. A malignant embolus might pass easily through the lymphatic trunk and be arrested in the smaller branches, and this appears to be the explanation of the condition found in this gland. The tissue of the mesocolon along the course of the lymphatic vessels showed no trace of growth. In this case, with a comparatively small growth in the colon, infection had already reached glands of the paracolic, intermediate and main groups, and the growth was most advanced in the intermediate gland. The glands had become affected by a process of embolism and not by permeation, as there was no sign of growth in the tissues of the mesocolon along the course of the lymphatic vessels (fig. 4).

It is instructive to note that Hartmann has drawn attention to the possibility of infection of the more distant glands. He writes: "In reality if one remembers that the lymphatic ganglia of this segment

are little developed, it will be understood that the neoplastic infection first manifests itself in the more important ganglia, which are situated on the arteries and at the origin of the arterial trunks in the retro-peritoneal tissue; the infection appears to pass over the regional ganglia" [6]. It is clear that the infection passes the regional glands because some of the vessels do.

In those portions of the colon supplied by long arterial arcades it is likely that the paracolic glands at some little distance from the seat of the disease may become affected; this applies in particular to the transverse colon, and to a lesser extent to the ascending and descending colon. The lymphatic vessels which emerge opposite the centre of the arcade have to travel in one direction or the other for a considerable distance parallel to the gut before they reach an arterial trunk along which they can pass towards the main groups. These vessels are likely to enter some paracolic gland on their way, while a vessel issuing opposite the trunk of one of the larger colic arteries would, after the same length of course, have reached an intermediate or even a main group gland. It is evidently necessary to remove the paracolic glands for some little distance on each side of the primary growth, and this, of course, necessitates excision of a considerable length of gut; in particular this applies to the transverse colon.

The ideal operation will consist in removing a considerable length of gut on each side of the growth, the primary glands, together with the vessels running to them from the gut, and the tissues in which these vessels lie—*i.e.*, the so-called "lymphatic area." As we shall show, if this is attempted it necessitates a great extension in the range of the operations usually practised, and may add something to the immediate risk. The added gain in a diminished likelihood of recurrence would appear to amply compensate for this increase in the risk. In some instances it seems likely that a wider excision of intestine will be attended with a lower mortality than a purely local excision.

A study of the course of the lymphatics shows that in some places it is impossible to remove the whole of the "lymphatic area"—for example, at the splenic flexure—and in these circumstances as much as possible must be removed. The question naturally arises, is it advisable to attempt the removal of all the secondary glands, particularly when the primary glands are malignant? In removing the primary glands one will at the same time remove many secondary glands, but it will be seen from the description given above how extensive is the secondary glandular system of any part of the colon, and how impossible it is to

excise the whole of this set of glands. In certain cases it may be found that, although the local conditions are favourable for excision, glandular hypertrophy has extended beyond the primary glands and has involved the secondary glands. If the secondary glands are certainly malignant, it would appear to be useless to proceed with the operation, except as a palliative measure. The difficulty of determining malignancy in any given enlarged gland must, however, be borne in mind, and where there is the least doubt the growth with the primary glands should be excised in the hope that the enlargement of the remaining glands may be due to absorption from the ulcerated surface in the bowel, and not

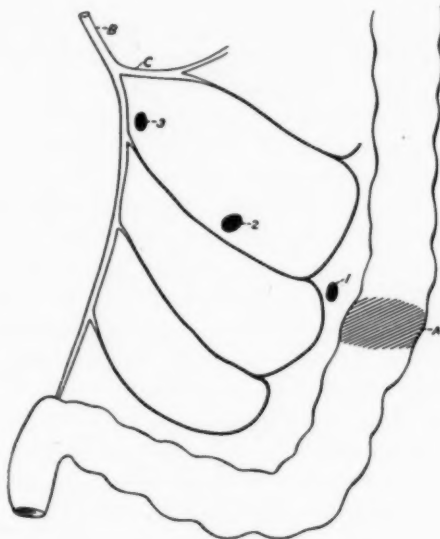


FIG. 4.

A, growth; B, inferior mesenteric artery; C, left colic artery; 1, paracolic gland; 2, intermediate gland; 3, main group gland.

to malignant infiltration. That is to say, in every case in which the local and general conditions are favourable the growth, with the corresponding "lymphatic area," or as much of it as is possible, should be excised, apart altogether from the presence or absence of enlargement of lymphatic glands.

We may now consider the possibility of excision of the "lymphatic

area" in connexion with a growth in the colon as it applies to the different portions of the colon in which such growths may be found. In the following pages we presume that the local conditions are favourable to excision, that there are no visceral or peritoneal metastases, or widespread gland disease. If the growth has given rise to marked obstruction, or from other causes, a palliative operation may have been performed previously—a typhlotomy, a colotomy, or an intestinal anastomosis; in such cases it may be necessary to modify to some extent the character of the radical operation.

The cæcum and lower part of the ascending colon have been dealt with in previous papers [7 and 8].

The upper part of the ascending colon, the hepatic flexure, and the first part of the transverse colon may be considered together (fig. 5). The primary lymphatic area corresponding to this portion of the colon comprises (1) the epiploic and paracolic glands; (2) the intermediate glands; (3) the main group glands of the middle colic chain. To remove the "lymphatic area" it will be necessary to tie the middle colic artery close to its origin, and to remove the mesocolon from this point up to the bowel. This will cut off the direct blood-supply to the upper part (possibly half) of the ascending colon and of about two-thirds of the transverse colon. It will then be necessary to excise the gut from the middle of the ascending colon to beyond the middle of the transverse colon, at about the junction of the middle and left thirds. But it will now be difficult to unite the ascending colon to the remaining portion of the transverse colon; this difficulty must be met either by closing both ends of the divided colon and effecting an anastomosis between the ileum and the sigmoid flexure, or by including in the part removed the lower part of the ascending colon, the cæcum and the terminal 6 in. of the ileum, and anastomosing the ileum to the remaining portion of the transverse colon. This would appear to be the better course. This procedure is evidently a formidable one, but it is doubtful whether it is more dangerous than a purely local excision of the hepatic flexure, with end-to-end anastomosis of the colon. The more extensive operation has some advantages; it permits free mobilization of the growth and complete control of the blood-supply, and enables the operator to bring the intestine out of the abdomen and to secure the field of operation from contamination when the gut is divided. Further, it allows an anastomosis between portions of intestine which are widely removed from the seat of disease, which are freely movable, and which can be brought together without tension.

The Middle of the Transverse Colon.—In the case of growth in this region a study of the lymphatics shows that all the vessels are intercepted by the paracolic glands before they have run very far. The intermediate and main groups do not receive vessels directly from the gut. Under these circumstances it would appear to be necessary to remove only as much of the colon on either side of the growth as will permit a satisfactory anastomosis, and in this way to secure those

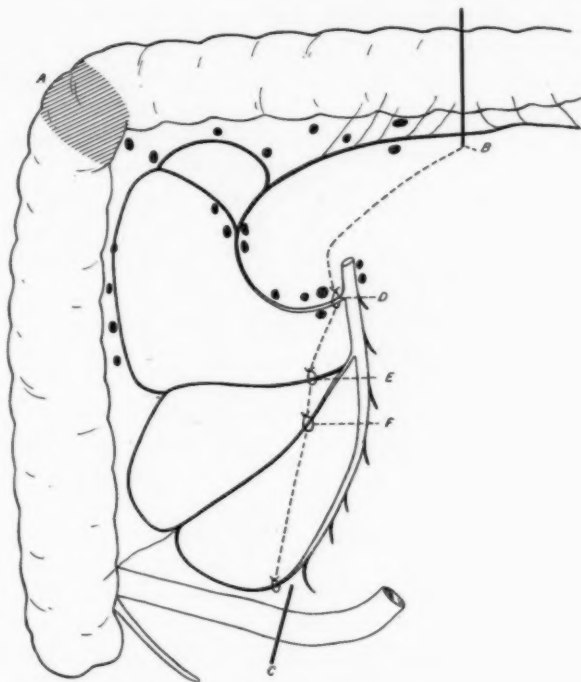


FIG. 5.

Excision of growth at the hepatic flexure. *A*, growth; *B*, line of section of transverse colon; *C*, line of section of ileum; *D*, point of ligation of middle colic artery; *E*, point of ligation of right colic artery; *F*, point of ligation of ileocolic artery.

paracolic glands at some little distance from the growth which might be affected (*vide supra*).

The Region of the Splenic Flexure (fig. 6).—It has been stated above that the large majority of the lymphatic vessels arising from this

part of the colon pass into the epicolic and paracolic glands, and that it is not common to see a vessel evading these glands and passing to one of the intermediate group. We have never seen a vessel passing directly to the main group of glands lying on the left colic artery or to the accessory group lying on the upper part of the inferior mesenteric vein as it runs above the duodenum, but we have drawn attention to the vessels which may sometimes be seen running directly to the splenic glands. The "lymphatic area" of this part of the bowel will include the epicolic and paracolic glands and the intermediate glands

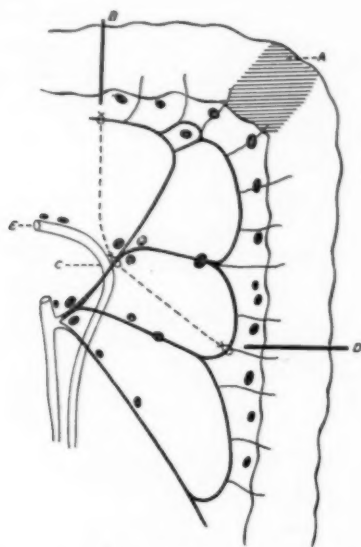


FIG. 6.

Excision of a growth in the splenic flexure. A, growth; B, line of section of transverse colon; C, point of ligature of left colic artery; D, line of section of descending colon; E, inferior mesenteric vein.

lying on the branches of the left colic artery, and also the glands at the hilum of the spleen. It is obvious that in performing excision for growths in the region of the splenic flexure it will be impossible to remove at the same time the "lymphatic area," as the splenic glands cannot be efficiently attacked. The surgeon must content himself with removing at the same time as the primary growth that portion of the "lymphatic area" attached to the branches of the left colic artery.

The operation is performed as follows: The left colic artery is defined as it leaves the inferior mesenteric vein and is tied at this point with the accompanying vein. The bowel is then freed by incising the peritoneum to the outer side of the ascending colon and splenic flexure and separating the bowel with the growth, the peritoneum and the sub-peritoneal tissues containing the lymphatic glands and vessels from the posterior abdominal wall. The peritoneum is then divided from the point of ligature of the artery upwards to a selected point on the transverse colon. The selection of this point will be determined by the extent of the transverse colon supplied by the middle colic artery; it will lie as a rule at the junction of the middle and left thirds of the transverse colon. The peritoneum is also divided in a direction downwards and outwards to the descending colon. At the selected point on the transverse colon the bowel is divided; below, the descending colon is divided in its lower part and the operation is completed by an end-to-end anastomosis. (See Case I, p. 173.)

The Descending Colon.—The "lymphatic area" corresponding to this part of the colon comprises the epicolic and paracolic glands and the intermediate glands on the branches of the left colic artery, including those on the first sigmoid artery. Some vessels also run to the splenic glands as in the case of the splenic flexure. It will then be necessary in removing this "lymphatic area" to tie the left colic artery at the point where it leaves the inferior mesenteric vein, and also the first sigmoid artery close to its origin. A reference to the diagram (fig. 7) will show that beyond this step the operation is much the same as that for the splenic flexure, except that, below, the gut will be divided in the first part of the sigmoid flexure.

The Sigmoid Flexure and the First Part of the Rectum.—We may consider the removal of malignant growths in this region as they occur (a) in the upper part and middle of the flexure; and (b) in the lowest part of the flexure and the first part of the rectum. It will be convenient if we first consider the second class (b) (fig. 8).

It may first be said that in the removal of growths in this situation, whether combined with excision of lymphatic glands or not, it is absolutely necessary to ligate the inferior mesenteric artery at some point; it runs too close to the growth to permit its preservation. The lymphatic glands receiving vessels from this portion of the bowel are the epicolic and paracolic glands lying close to the gut and the intermediate and main groups represented by the glands around the inferior mesenteric artery. We have not seen direct vessels entering glands at a higher

level than the junction of the middle and upper thirds of the artery, well below the point of origin of the left colic artery. The apex of the wedge of tissue to be removed will, therefore, lie on the inferior mesenteric artery just below the left colic. The base is formed by the gut, and the intervening tissue will include the inferior mesenteric artery, the lower sigmoid arteries, the glands named above, the lymphatic vessels, and the subperitoneal tissue in which they lie. To determine the amount of gut to be removed we are guided, so far as the upper limit is concerned,

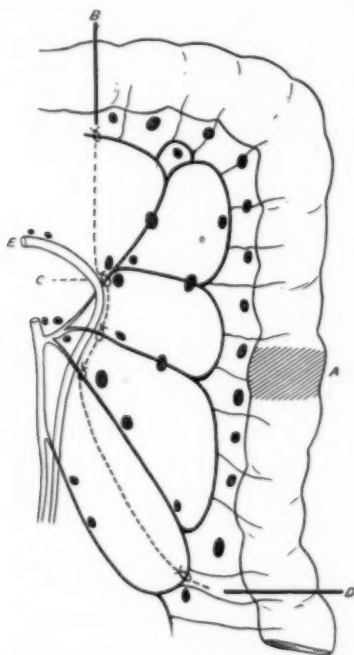


FIG. 7.

Excision of a growth in the descending colon. *A*, growth; *B*, line of section of transverse colon; *C*, point of ligation of left colic artery; *D*, line of section of sigmoid flexure; *E*, inferior mesenteric vein.

solely by the necessity of going well above the growth, for, the left colic artery being intact, the blood-supply of the upper part of the sigmoid is assured because of the free anastomoses between the left colic and sigmoid branches. Mr. Moynihan [12] considers it essential to remove

"a gland," which he describes lying on the trunk of the inferior mesenteric artery above the origin of the left colic; this is to be done either by stripping the gland off the arterial trunk when possible, or by ligaturing the artery at its origin. We do not know on what ground Mr. Moynihan attaches such importance to the removal of this particular gland, as he adduces no evidence, anatomical or pathological, in support of his view. A reference to the diagram (fig. 2) based on our specimens will show that the glands in this situation are secondary, and that there is no more reason for interfering with them than with the lower lumbar glands. Moreover, ligature of the trunk of the inferior mesenteric artery at its origin

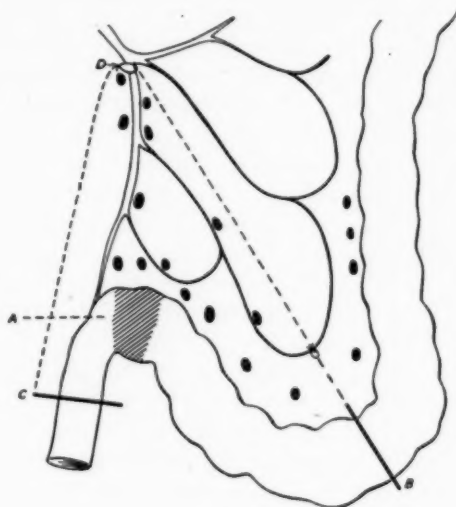


FIG. 8.

Excision of a growth at the junction of the sigmoid flexure and the rectum. *A*, growth; *B*, line of section through the sigmoid flexure; *C*, line of section through the rectum; *D*, point of ligature of the inferior mesenteric artery.

endangers the blood-supply of the upper segment of the bowel; it will then depend entirely on the single anastomosis between the middle and left colic arteries. It is true that it is possible in the dead body to force injection material from the middle colic artery into the sigmoid arteries after the inferior mesenteric artery has been tied at its origin; also it is possible for the circulation of the gut to be maintained in animals after the artery has been tied—Archibald [1], Okinczyc [13]. But

these observations do not tell us whether we may rely on the anastomotic circulation in living human beings; it may be so in comparatively young and healthy individuals, but probably not in elderly subjects afflicted perhaps with diseased vessels and a feeble circulation. It may be that the connexion between the middle colic and inferior mesenteric arteries is physiologically inadequate. Mr. Moynihan advises that the efficiency of the blood-supply to the upper cut end of the bowel may be tested by releasing the intestinal clamp to see if it bleeds; but what is to be done if it does not bleed?

The lower limit of the excision is determined not by the simple necessity of going well below the growth, but by a consideration of the blood-supply. The vitality of the lower segment will depend on the middle and inferior hæmorrhoidal arteries and branches of the sacral arteries. We have not yet enough evidence that these are sufficient in all cases to permit the retention of a length of the rectum necessary for the performance of an anastomosis.

Briefly, the operation consists in exposing the inferior mesenteric artery and ligaturing it and the vein just below the point of origin of the left colic artery. A long incision through the peritoneum is made on the outer side of the mesorectum and mesosigmoid, and the descending colon and the gut mobilized by stripping it inwards towards the middle line. In doing this the ureter and the spermatic vessels will be encountered and must be avoided. The mesosigmoid is then divided in an oblique line downwards from the point of ligature of the inferior mesenteric to about the middle of the sigmoid flexure, care being taken to preserve as far as possible the secondary arches on the sigmoid arteries. The peritoneum to the inner side of the artery is then divided downwards to the inner side of the mesorectum. The mass of tissue to be removed is then stripped forwards from the upper part of the sacral hollow, and the gut clamped and divided at the middle of the sigmoid flexure and at the junction of the first and second parts of the rectum. The part removed will consist of the lower half of the sigmoid flexure and the upper part of the rectum, the greater part of the inferior mesenteric vessels with the accompanying chain of glands and lymph-vessels, and the surrounding subperitoneal tissues.

The blood-supply of the upper end of the bowel is assured owing to the preservation of the left colic artery; that of the lower end depends, as we have said, on the middle and inferior hæmorrhoidal arteries, and is suspect. It must be tested by releasing the clasp to see if the cut edge of the rectum bleeds freely. If it does, and other conditions are

favourable, the operation may be terminated by an anastomosis. To do this and to bring the sigmoid flexure down without tension it may be necessary to further mobilize the descending colon and even the splenic flexure. If the blood-supply of the lower segment is insufficient two alternatives present themselves. The first is colostomy, which will probably be necessary in a considerable number of cases. The second consists in the removal of more of the rectum, even down to the levator ani, and the completion of an anastomosis by everting the rectum through the anus, pulling down the sigmoid flexure out through the everted rectum and uniting them below by Maunsell's method. There can be no doubt that this operation, however "ideal," is one of great severity, and in any but absolutely favourable cases is likely to be terminated by a permanent colostomy. In cases where a colostomy has been done previously on account of obstruction, although it may be possible to close the opening and effect an anastomosis by the method described, it will probably be wisdom to abandon this and leave the patient with a permanent artificial anus. (See Case II, p. 173.)

(a) Growths in the Middle and Upper Part of the Sigmoid Flexure.

The lymphatic glands receiving direct vessels from this part of the colon include the epicolic and paracolic glands, the intermediate glands lying on the sigmoid arteries in the mesosigmoid, and the main group glands on the inferior mesenteric artery. The "ideal" operation in these cases is practically the same as that described under group *b*, with the difference that more of the mesosigmoid will be removed and the gut will be divided above at the junction of the descending colon and the sigmoid flexure. It seems likely, as we have said, that the "ideal" operation will be terminated in a large proportion of cases by a permanent colostomy, and it must be remembered that the purely local excision of growths in this situation, with preservation of the alimentary circulation, have been attended with a considerable measure of success. If the "ideal" operation necessitates a colostomy the comparative disadvantage is so enormous that it will rarely be performed. Lacking as we do sufficient knowledge as to the possibility of anastomosis after excision of the inferior mesenteric trunk, and being unwilling to consider even the possibility of a permanent colostomy after excision of a growth in the middle of a sigmoid flexure, we must perform an operation which, if short of the ideal, does remove the majority of the possibly affected glands. The operation (fig. 9) consists in exposing the inferior mesenteric vessels, dividing the peritoneum over them, and stripping off as

many of the glands of the main group as is possible without damaging the vessels. This is done from the point of origin of the left colic artery down to the origin of the lowest sigmoid artery. The sigmoid arteries are tied at their origin, the lowest being preserved if its removal be not necessitated by the position of the growth. The descending colon is then mobilized and the gut divided above at the junction of the descending colon and the flexure, and below in the lower part of the flexure. Almost the whole of the mesosigmoid will thus be

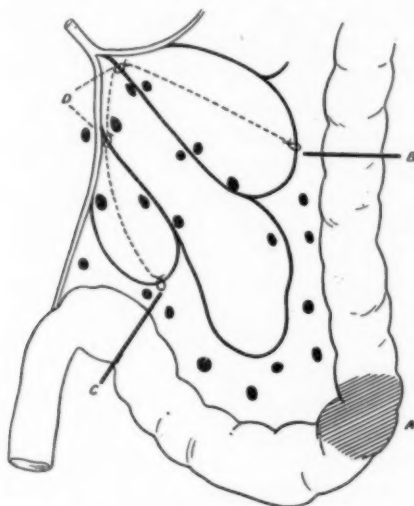


FIG. 9.

Excision of a growth in the middle of the sigmoid flexure. *A*, growth; *B*, line of section above the growth; *C*, line of section below the growth; *D*, the sigmoid arteries tied at their origin.

removed, including the intermediate, paracolic, and epicolic glands, and probably the greater number of the main group glands corresponding to this part of the sigmoid flexure. The operation is terminated by an anastomosis between the end of the descending colon and the lowest part of the sigmoid flexure. If it is found necessary to tie the lowest sigmoid artery at its origin care must be taken to divide the gut below the level of the brim of the pelvis, in order to avoid the "dead end" which may be left, as Manasse and Archibald have shown, owing to the non-union of the branches of the lowest sigmoid and the superior hæmorrhoidal arteries. (See Case III, p. 173.)

Case I.—A man, aged 53, was admitted to the Leeds General Infirmary under the care of Dr. Maxwell Telling on October 31, 1908, suffering from acute intestinal obstruction. The cæcum was found to be enormously distended, the muscular coat cracking at the slightest touch, and a small annular growth was found at the splenic flexure. A Paul's tube was tied into the cæcum, a considerable part of which was pulled out of the abdominal wound owing to its damaged condition. The patient was extremely ill for some days, but eventually recovered. On December 9 the growth was excised by the method described above. He made a straightforward recovery from the operation, but the typhlotomy opening is not yet closed and will probably require suture.

Case II.—A man, aged 32, was admitted to the Leeds General Infirmary on July 26, 1907, suffering from intestinal obstruction. A growth was found in the lower part of the sigmoid flexure, and an iliac colostomy was performed. (It should be said that he came under the care of one of us in the absence of Mr. Moynihan.) On September 4, 1907, the growth was removed by the method described, the colostomy being left as a permanent artificial anus. A coil of small intestine was adherent to the median exploratory incision made at the first operation, and was so much kinked that an anastomosis was performed between the intestine above and below. His recovery was delayed by the formation of a pelvic abscess which discharged into the stump of rectum remaining, but he returned home in good health on October 26.

Case III.—A woman, aged 54, was admitted to the Leeds General Infirmary on December 11, 1908, suffering from intestinal obstruction of five days' duration. The abdomen was explored through a median incision and a growth found in the middle of the sigmoid flexure. Typhlotomy was performed. On December 31 the growth in the sigmoid was excised by the method described above and an end-to-end anastomosis effected with ease. In stripping forward the subperitoneal tissues the left ovarian veins were injured, so the ovary was removed. A subserous uterine myoma was also excised, the uterine wound being closed by catgut sutures. She has made a satisfactory recovery, though the typhlotomy wound is not yet quite closed.

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The PRESIDENT (Mr. Warrington Haward) said that the members of the Section would wish to accord their best thanks to Mr. Dobson and Mr. Jamieson. When surgeons reflected how the successful removal of malignant diseases depended upon the removal also of any affected lymphatic area, they must see that such an anatomical demonstration as the paper afforded was of the greatest surgical value. They were further indebted to Mr. Dobson for bringing to their notice the series of very beautiful specimens which were on the table. He thought it possible that a paper of this kind, dealing with facts rather than with theories, did not admit of very much discussion.

The Torus Palatinus.

By RICKMAN JOHN GODLEE, M.S.

IN May of last year Dr. Morrision Davies brought to see me a lady aged 58, a Jewess, on account of a small projection, obviously of bone, terminating posteriorly a median ridge in the hard palate, which, she said, had been present for a long time, but had slowly increased in size (fig. 1). The most prominent part corresponded approximately to the

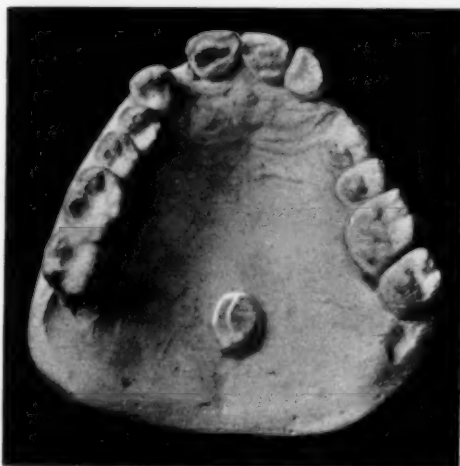


FIG. 1.

junction of the palate bones and superior maxillæ. The mucous membrane on its surface presented a small healing ulcer, and the question of possible malignancy was the actual cause of the consultation.

Up to that time I had never heard of the *torus palatinus*, and, surgeon-like, I suggested the removal of the supposed exostosis. But my ardour was damped by the deaf husband mistaking my suggestion that it was congenital for one that it was hereditary, and stating in confirmation that her daughters had the same peculiarity. We at once

examined his palate and our own and found them to be flat, while that of the grown-up daughter who accompanied them presented the well-marked median ridge, the model of which I produce. The husband's statement turned out to be an exaggeration. Dr. Davies has examined all the other children, and several if not all the grandchildren, and in none of them could he discover any abnormality of the palate.

In October I was operating on an American, aged 69, for piles, and the anæsthetist, in searching for the possible presence of artificial teeth, discovered a torus almost exactly like the one I have described, only situated somewhat further forward (fig. 2), being between the first (?)



FIG. 2.

molars instead of well behind the second (?), the anterior part being $\frac{1}{4}$ in. nearer the incisor teeth than in the last case. The patient, a very intelligent man, was unaware of the presence of the lump, though he did recollect that it had been remarked upon by some dentist or doctor. He has promised to examine his children and grandchildren, but has not yet reported.

In July Dr. Batty Shaw sent me a middle-aged woman from the out-patients' department with the flat swelling of which I now show the cast. It extends up to the alveolar border in front, and posteriorly as far as the interval between the second and third molars. This ridge has a well-marked sagittal groove in the middle line.

Naturally I made inquiries amongst the dentists, and it at once became clear that to some of them the swelling was familiar, as, if it be at all common, was likely to be the case, for it must necessitate modifications from the normal shape of an upper palate. Some of my friends had preserved casts of palates showing the peculiarity; Mr. William Hern has kindly given me five and Mr. Relph one. Mr. Tomes had gone so far as to attempt to remove one. But it was humiliating when Mr. Mummery produced the cast of the largest torus I have ever seen (fig. 3), and told me that he had submitted it, or the patient (an American lady) from whom it was taken, to me six years

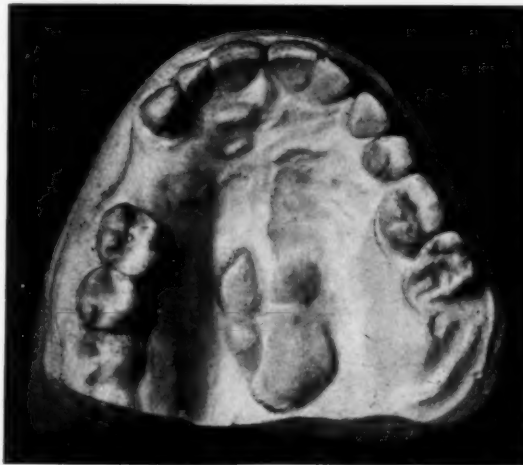


FIG. 3.

ago, and that we agreed we knew nothing about it and decided that it should be watched. She is still a patient of Mr. Mummery, and the lump has apparently very slightly increased in size posteriorly, possibly owing to some thickening of the soft parts. The swelling in this case is not quite symmetrical, but presents an extra lobe on the right side. The median groove is well marked. It extends as far forward as the alveolar border and backward as far as the back of the third molars. Mr. Mummery has discovered one other cast of a long flat torus.

One of Mr. Hern's cases shows a torus that approaches the last in size and extent (fig. 4), but it is quite symmetrical. It was taken eight

years ago from an English lady then aged about 40. The lump had been recognized for two or three years, and it has been seen since the cast was taken. It does not appear to have grown. Another of Mr. Hern's casts shows a well-marked posteriorly situated swelling, prolonged forward by a flat ridge, which therefore resembles my first case (fig. 5). The abrupt part of the tumour has a median groove. It was taken a year ago from an Englishwoman aged about 40. His third cast shows a small abrupt torus opposite the first molars (fig. 6). It is peculiar in that it is just to the left of the median line. But I think it is the genuine thing—as I have found at least two lateral tori

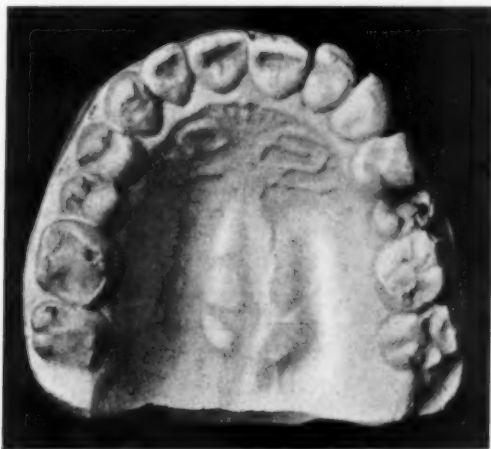


FIG. 4.

amongst the skulls at the College of Surgeons. The cast was taken about five years ago from an Englishman aged about 60. On comparing it with a cast taken recently it is clear that it has not grown. There is no sign of a similar growth in his four children, who are now of adult age. His fourth cast shows a large posterior torus markedly bilobular, the left lobe being the larger. It is opposite the space between the second and third molars. It was taken recently from an Englishwoman aged about 50. She knows of no other cases in her family; but she was unaware that she herself had any peculiarity. His fifth is situated far forwards. It is flat and made up of four lobes.

It was taken from an Englishwoman aged about 45, and has apparently not increased in size during the last five years.

Mr. Relph has given me a cast almost exactly like that of the third of my own cases: a flat ridge with a median groove. It was taken from an Englishwoman some years ago.

Such a collection of odd-looking palates made in little more than six months was sufficiently interesting, and it became necessary to inquire whether the swelling was pathological or anatomical. Dr. Keith, the learned curator of the Hunterian Museum, at once referred me to the exhaustive monograph by Stieda, of Königsberg, "*Der Gaumenwulst*"

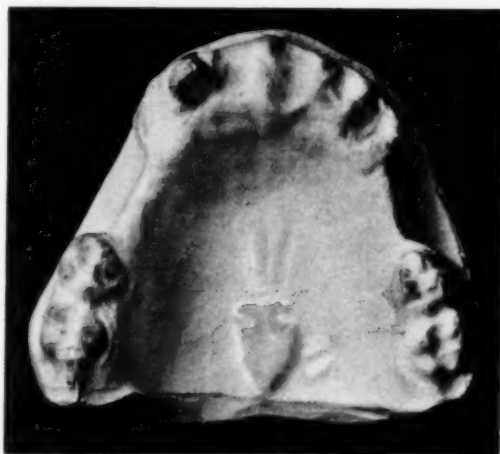


FIG. 5.

(*torus palatinus*) in *Virchow's Festschrift*, 1891, i, p. 145. The whole subject is there treated with much detail. Chassaignac seems to have been the first to draw attention to it and to have attributed the swelling to syphilis, which it certainly is not. Luschka, Virchow and others briefly refer to it. But it was not seriously studied till 1879, when Kupffer and Bessel-Hagen started the theory that it was a characteristic of Prussian skulls. This theory has been completely upset by later and more extensive observations. Stieda concludes that it is met with in the skulls of all races, though it has actually been observed, he says, most frequently in those of Peruvians and Ainos, and least often amongst

negroes. Amongst 229 Peruvian skulls it was found in 129 cases—that is, in 56.3 per cent.—and amongst sixty skulls of Eskimos it occurred in no fewer than 60 per cent.

In at all events one anatomical book¹ it is described and figured as an anatomical variation. He figures (fig. 7) three frontal sections of common varieties of hard palate, one (*a*) very thin with a slight median groove on the under surface and flat nasal surface; another (*b*) thicker, quite flat beneath, but with the nasal surface somewhat concave from side to side, and a third (*c*) with a rough buccal surface, very thick, and with a much hollowed nasal surface. It is not, therefore, surprising in handling a large number of skulls to meet with great variations. The

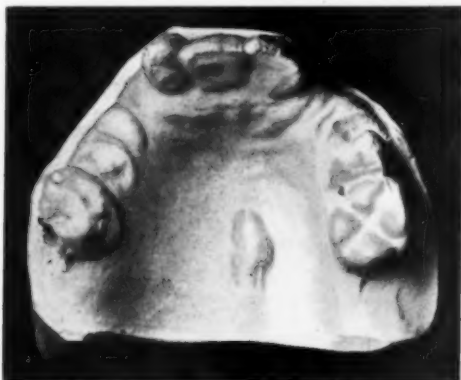


FIG. 6.

book description of the palate of an Anglo-Saxon or a German would need considerable editing before it would apply to that of a Peruvian, an Eskimo, or a negro. Not to dwell upon such striking features as the variations in depth of the alveolar process giving rise to a high or a low palate, or in the shape of the curve of this process which varies from that of a hyperbola to that of a horseshoe, there are some of apparently minor importance which may give rise to or modify the appearance of a torus palatinus.

The palate process of the palate bone is sometimes (often, *e.g.*, in the skulls of Hindoos) extremely smooth and thin, quite translucent, in fact,

¹ "Raubers' Lehrbuch der Anatomie des Menschen," 1906, Ab. ii., pp. 294—299.

showing but slight indications of the crest, which, running inwards between the two posterior palatine canals, serves for the attachment of a part of the tendon of the tensor palati, and is quite plain near the middle line. In some skulls, on the other hand, these crests form great prominent wings. In other skulls there is a deep hollow varying much in depth and sharpness of outline, which lodges a mass of glandular tissue. If well marked the two hollows, one on each side, and running into one another in the middle line, appear to form the termination of a median ridge, even if there be no true *torus palatinus* (fig. 8). Again, the large posterior palatine foramen, instead of being a round opening or a narrow slit flush with the under surface of the palate bone, may open out into a wide trumpet-shaped orifice; and if the two grooves which lodge the

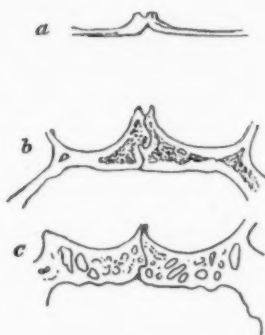


FIG. 7.

vessels are deep, the whole of the posterior half of the palate becomes convex, and resembles to some extent the wider forms of torus, if, indeed, it does not actually form one (fig. 9).

In a few cases I have met with a palate completely occupied by two equal rounded elevations running from front to back, separated by a deep median groove. In them the palate is altogether thin, and the floors of the nasal fossæ are hollowed to a corresponding extent—much more so than is shown in Rauber's figures. This is a totally different thing from that which we are investigating (fig. 10). Occasionally the whole palate is like an inverted pent-house roof with a ridge along the middle line. Such a palate is thicker towards the middle than at the sides (fig. 11). Sometimes the whole palate is thin and arched from side to side (fig. 12). Lastly, it must be observed that while the palate

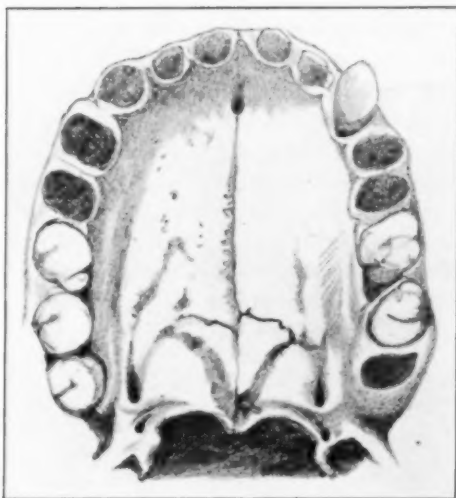


FIG. 8.

Indian, No. 669. Not a true torus, but very well-marked hollows for adenoid tissue and marked prominence between them; slit-like openings of p.p. canals.

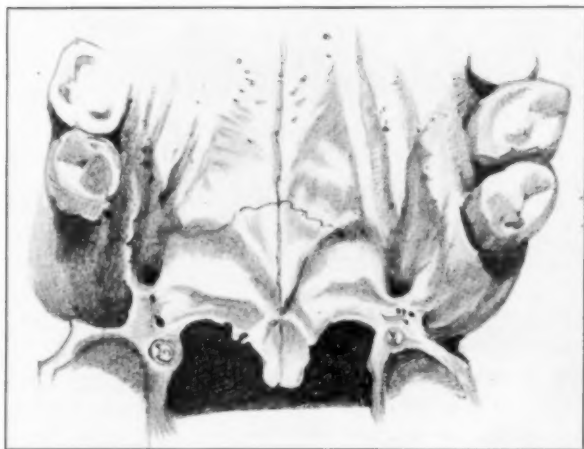


FIG. 9.

Polynesian, 1077. Very deep vascular groove, sharp ridge between glandular fossae, very wide palate, processes of palate bones.

in some races is usually smooth and even, in others it is almost always rough and irregular, deeply scored by grooves and foramina for vessels. Very often a more or less deep groove marks the mesial suture, which is, however, as often the seat of a narrow ridge, and it is common to find the ridge and the groove together.



FIG. 10.

Congo, 1255, 67. Double convex palate, concave floors of nasal fossae.

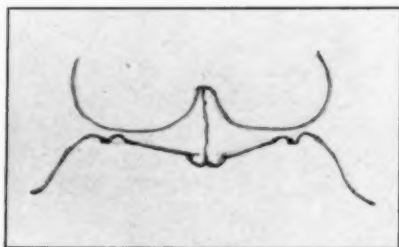


FIG. 11.

Japanese, 719. Penthouse roof, deep vascular grooves.

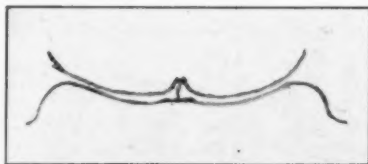


FIG. 12

Indian, 690, 6. Thin, convex palate, with corresponding concave floor of nose.

The last observation leads up to a description of what is actually meant by the *torus palatinus*. As most commonly seen, the *torus* (Latin *tōrus*, which means any kind of lump from a brawny arm to a marriage

bed) consists of a more or less narrow ridge corresponding to the median suture of the palate, extending forwards to the anterior palatine foramen and backwards well on to the palate bones. In this form (fig. 13) it tapers gradually in front, but more abruptly behind opposite the glandular fossæ on the palate bones. The ridge is often, however, much wider, and may occupy as much as two-thirds of the whole width of the palate (figs. 14 and 15). The amount of projection varies greatly. Sometimes, instead of a uniform smooth ridge, it is made up of irregular rounded bosses (fig. 3), and, as shown in some of my casts (figs. 1 and 2), it may happen that one part only is raised into a prominent tumour

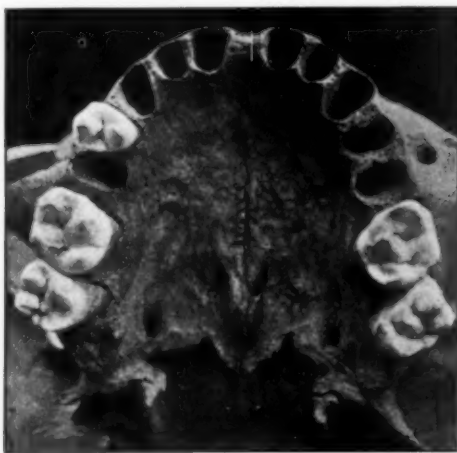


FIG. 13.

Large posterior torus prolonged forwards by narrow ridge (Whitechapel skull).

which would strike the eye of the most casual observer. The most irregularly bossy *skull* I have seen was found in an early Saxon chalk coffin at Chertsey Abbey (fig. 16) (Royal College of Surgeons Museum). The median groove is often deep; and if the projections are wide and flat, it is a sulcus rather than a torus which catches the eye.

The torus consists of spongy bone, as may be seen in sections. It is not produced by a bending downwards of the palatine processes, the floors of the nasal fossæ remaining either flat or only hollowed to a normal extent. It is really essentially a projection downwards of the *diplœ*, though the compact tissue on the surface varies very much in thickness, as is shown



FIG. 14.

Torus reaching the whole length of the palate (Whitechapel skull).



FIG. 15.

Very long and wide torus, race unknown. (University College Museum.)

in the two sections exhibited (figs. 17 and 18). I believe that the variety which causes the marked single prominence shown in several of my casts of living palates is generally the posterior extremity before referred to as existing between the two glandular fossæ. But it is remarkable that though I have so many of these in casts taken from the living body I have not often found it in the skull. The best specimen I have seen is in one of the longbarrow skulls at the Oxford University Museum.

This description is the result of the examination of a large number of skulls of different nations to which I was led by Stieda's statements in his monograph with regard to the occurrence of the torus in greater or larger



FIG. 16.

Skull from an Early Saxon burial. (Hunterian Museum.)

proportions or development in all races of which a sufficient number are examined. It is evidently a mistake to attempt to estimate the percentage of skulls from any particular race which show indications of its presence. The passage from the normal to the abnormal is so gradual that different observers, or even the same observer on different days, might arrive at different results. The observations are tabulated, and it will be seen that whilst there are some agreements with Stieda's statements there are some curious differences—for example, amongst the ninety-seven Peruvian skulls at the College of Surgeons, I only found eight very slight attempts at a torus, whereas Stieda says it occurs as

often as 56.3 per cent. Again, our seven Aino skulls show hardly a sign of it, though he says it is common amongst them. I have found it well marked and very common amongst the Tasmanians; well marked but less frequent amongst the Australians; well marked and frequent amongst the Fuegians, though not common amongst the Patagonians and South American Indians; very common among the Polynesians, especially some New Zealanders, but not common amongst the Melanesians; common but badly marked amongst the Eskimos, Lapps

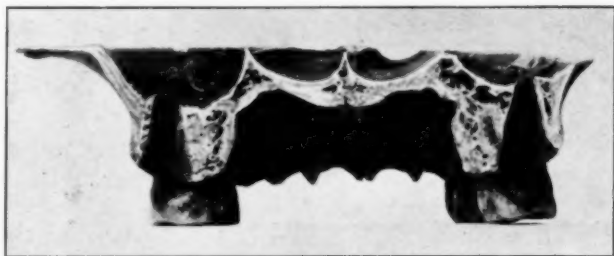


FIG. 17.

Section of torus of moderate size; thickening of lower table as well as diploe.

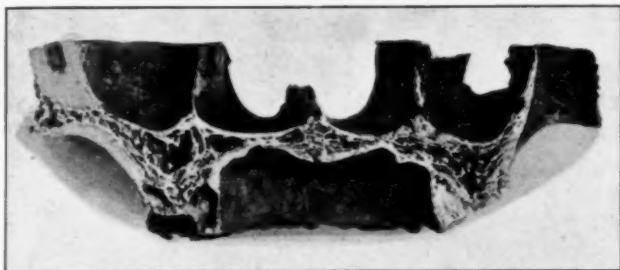


FIG. 18.

Section of moderate-sized torus, showing that it is caused by a thickening of the diploe.

and Finns; uncommon amongst the Hindoos and other inhabitants of India; never amongst the Veddass, very rare indeed amongst the negroes and Hottentots, and moderately frequent amongst the various European races and the North American Indians. There are hardly any among our large collection of Peruvian skulls.

The longbarrow skulls are very remarkable. They have often very rough palates, and a large proportion have the torus well developed. One is shown in fig. 19.

I saw about sixty skulls of lunatics at Oxford, about ten of which had a more or less well-developed torus—not a very large proportion. They came, I believe, from a military lunatic asylum.

I have reproduced, slightly modified, on a diagram, Deniker's grouping of the human races according to their affinities. And I have placed a dotted line under the names of the races in which, according to



FIG. 19.

Longbarrow skull at Oxford, No. 22. Showing rough palate, very large posterior torus, very deep median groove, large crests, and only one canal on each side.

my own experience, the torus is very common, and a black line under that of those where it is practically unknown. The names of the races in which it occurs with moderate frequency are unmarked. I am not an anthropologist, and I do not know how far Deniker's scheme is accepted, but the grouping of the lines is at least remarkable. It looks rather as if the torus is a mark of the primitive races which, like the

special Alpine flora, have been pushed to the limits of civilization and continue there to thrive under circumstances which are not suitable for their advanced descendants.

In connexion with this branch of the subject I may refer to two interesting series of skulls presented to the museum of University College by Professor Thane. They were found in two pits, supposed to be plague pits—one in Liverpool Street and the other in White-chapel. Many details with regard to these skulls have been put on record in *Biometrika*, iii, Nos. 2 and 3, March and July, 1904, and v, Parts I and II, October, 1906, by R. W. Macdonell, LL.D. The observations were made under the direction of Professor Karl Pearson, and these gentlemen very carefully investigated the history of the localities, and concluded that the pits were probably plague pits. If so, the burials were those of East End or City Londoners of 200 years ago. One hundred and seventy-six of those skulls have palates, and amongst them nineteen present good examples of the various degrees of development of the torus—a rather high percentage, if we are to talk of percentages. But East End Londoners were probably then as now a mixed breed.

It is not strange that one should meet with a series of skulls of a particular race in which more or less of this prominence is the rule. Such, for example, is the case with our Polynesian skulls at the College. Indeed, in a collection of twenty obtained from the museum at Auckland, New Zealand, almost every skull has a well-marked torus. Nor is it surprising that amongst other races, like the negroes and their allies, it is practically never met with. But it is remarkable that amongst people like the Hindoos, whose palates are usually smooth, a pretty well-marked torus should occasionally be seen. The fact that amongst the very mixed peoples of Europe it occurs with very varying frequency is consistent with its being a racial peculiarity, and of this there seems to me to be no reasonable doubt.

It may be asked why a paper should be offered to the Surgical Section on an anatomical peculiarity which has certainly been recognized for thirty years; and, indeed, as far as mere anatomical facts are included in it, some apology no doubt is due to the Section. But the answer is that the existence of the condition is apparently not widely recognized by English surgeons, and, as far as I know, no mention is made of the *torus* in our surgical text-books. I think it is high time that it took its proper place amongst tumours of the palate, and that the warning were issued that no surgical treatment is required. Probably it is best to

retain for it the name which anatomists have given it. It is pretty clear that it is not a pathological condition, for it appears to have been observed in the foetus; the only known pathological condition it at all suggests is leontiasis ossea, and that only because it is a swelling formed of spongy bone occurring in the maxilla, but the resemblance ends here, for having reached a certain size it appears to remain almost if not quite stationary.

Comparative anatomy does not help us much, if at all. It does not occur amongst anthropoids, but it is said to have been seen in cynocephalus babuin. The baboons, some of them certainly, have a very marked ridge, but a sharp one in the middle line, and in one of our skulls at the College of Surgeons is a swelling that might be called a torus, but it is one of a rickety animal from the Zoological Gardens, and so just open to question. The gorillas and chimpanzees and lemurs and other monkeys in the collection have perfectly smooth palates: much smoother and whiter than those of the Congo skulls. Mr. Turner tells me of well-marked ridges he has observed in some of the lower animals, and I have seen some myself, but have not worked enough at this branch of the subject to make any definite statement.

The only new facts that I have to add to those of former observers are:—

(1) That it has been met with once, and almost certainly twice, in two members of the same family.

(2) That in certain adult patients who have been under observation for a considerable time the tumour has not appeared to increase in size.

(3) That the swelling is sometimes so abrupt at one part as to make the ridge escape observation and suggest the possibility of the torus being a localized exostosis. This is, I think, important, as it is just these cases that invite removal by an operation.

RACES OF MANKIND ARRANGED ACCORDING TO THEIR AFFINITIES (DENIKER).

Race	No. of skulls examined	Occurrence of torus	Remarks
I—Bushmen ...	27	0	The palates are rough and the vascular grooves deep
II—Melanesian ...	110	0	New Caledonia, New Hebrides, Tokelau, Evandunga, &c.; two skulls showed narrow central ridges, but no true torus
Papuaus ...	40	0	One had a narrow ridge
Negroes and West Africans and Central Africa ...	184	0	Palates long, square, like a gorilla's, arch high, very rough, massive; there are no cases of true torus
Congo ...	76	0	One a median ridge in front; one a median ridge behind; palates usually flat
Madagascar ...	6	0	Rough palates
III—Ethiopian ...	—	—	—
IV—Australian ...	116	17	They often have a rather well-marked prominence behind
Tasmanian ...	37	17	Many of the other skulls show a tendency to the torus—in fact, most of them
V—Dravidian ...	—	—	—
Veddahs ...	25	0	—
VI—Assyroid ...	—	—	—
VII—Indo-Afghan ...	528	22	Includes Hindus, Afghans, Pathans, Cingalese, which I cannot analyse; the palates are often high, and often smooth; the torus is sometimes well marked, though seldom large
VIII—North African			
Algiers ...	22	4	All good examples
Guanches ...	33	8	Four are doubtful
Egyptian ...	145	2	Very poor ones
IX. Melanochroid)			(These follow in sequence as arranged in the museum
X. Xanthochroid)			(of the Royal College of Surgeons
Hanoverians ...	2	0	—
Hessians ...	2	0	—
Prussians ...	16	3	Some others had a slight ridge which would not have attracted attention if they had not been Prussians
Saxon ...	1	0	—
Baden ...	1	0	—
Swiss ...	2	1	—
Czech ...	3	0	—
Poles ...	5	1	—
Croats ...	2	0	—
Magyars ...	3	1	—
Hungarian ...	1	0	—
Slovak ...	1	0	—
Russmark ...	1	0	—
Gipsies ...	7	2	—
Roumanian ...	7	0	—
Bulgarian ...	2	1	—
Russians ...	11	2	—
Cossacks ...	5	1	—
Danes ...	1	0	—
Swedes ...	12	1	—
Dalecarlian ...	1	0	—
Anglo-Saxon in historical times ...	146	16	—
London plague pits ...	176	19	—
Total ...	408	48	—
English pre-historic ...	40	20	These comprise 13 skulls at the Royal College of Surgeons and 27 at the University Museum, Oxford; although I have only put down 20 as having a torus, 9 others were either very rough or showed an indication of a torus; 11 only were smooth

N.B.—The Fuegians and Tasmanians are added to Deniker's table.

DISCUSSION.

The PRESIDENT (Mr. Warrington Haward) said that the Section was indebted to Mr. Godlee for his interesting communication. He had done good service in bringing this anatomical fact to their notice, and, after all, anatomy and surgery were not very easily divided. He (Mr. Haward) thought that possibly several of the members—he knew that it was so in his own case—had been misled into thinking that the torus was a pathological condition. He recently saw a lady who had this bony swelling on the palate, and when the question of its removal was submitted to him, he, with a caution that was perhaps significant of advancing years, advised that as it caused no inconvenience, and had existed on the palate for a considerable time, it might be left alone. He wished, however, to confess his ignorance of the fact that the supposed growth was one of these anatomical peculiarities.

Mr. BUTLIN said that when he first saw the title of Mr. Godlee's paper he wondered what it could be about. "Taurus" meant a bull, and "Palatine" seemed to refer to Rome, so that the subject suggested to him a Bull of Excommunication. He had seen one of these cases—that of a lady 50 or 60 years of age, who was brought to him two years ago. He had never previously seen a case, and he did not know what the name of it was, or any facts about it. The tumour was not quite symmetrical, and there was that curious groove between the two portions of it of which mention had been made in Mr. Godlee's paper. He came to the conclusion that it was probably not a new growth, and, indeed, he had a suspicion that it was congenital. The lady, however, was quite sure that she had had it only for a short time, but he found the real fact to be that her attention had only recently been called to it by biting on a nut or a piece of hard bread. This caused a bruise, and drew her attention to what she thought to be a new growth in the palate. He was greatly tempted to take it out, but instead of doing so he suggested that she should have a mould made, and a plate fitted so as to protect it from injury. At the end of six months another mould was made, and the two compared, so that any difference in the size and shape might be noted. The swelling on this comparison appeared to be very slightly larger than before, although the two portions seemed to retain their relation one to the other. He was glad he had not removed it, particularly since hearing the very lucid explanation they had had from Mr. Godlee as to the probable nature of this condition. He had a suspicion that the small increase of size might be accounted for by superficial thickening left by periostitis.

Dr. ARTHUR KEITH said that so far as his knowledge of the matter went he owed it all to Mr. Godlee. He was interested in the anthropological side of the question, and he pointed out that the races which Mr. Godlee had mentioned as showing the torus in greatest frequency were closely allied to

one another. It had often been asserted, for instance, that the Lapps were most nearly related to the ancient Europeans, and there were other peoples figuring in Mr. Godlee's list which many authorities believed to have a close racial relationship. This bore out Mr. Godlee's inference that the presence of the torus was concerned in some way with racial characteristics. He (Dr. Keith) had had to do with one of these cases, and he had also noticed the peculiar appearance in various skulls, but as he did not understand it he did not inquire further. They got into the habit of not going on with things they did not understand, and thereby they missed a great deal. The torus appeared on each side of the median suture of the palate, and was evidently of the same nature as those sutural ridges or outgrowths found along the sutural lines on the roof of the skull—especially along the interfrontal and sagittal sutures—of Australian and other primitive races. In many cases this development of bone did not take place until later in life. The sagittal suture was well seen in many of the Australian skulls.

Mr. J. G. TURNER said that he had looked into the subject of the torus palatinus two years ago, being attracted by a statement in the text-books that it was a stigma of degeneration. As he was at that time looking through asylums with a view to determining what was the value of the high dental arch, he took the opportunity also to look for the torus palatinus. Out of 530 inmates of asylums he found only two instances of the presence of the torus, and he considered that this effectually disposed of the idea that it was a stigma of degeneration. He found two other examples in normal patients at the Royal Dental Hospital. He had also found examples in the skulls of other races, the best marked being in an Eskimo skull. One of his cases furnished an example of a small torus occurring far back at the junction of the palate processes of the maxillæ with the palate bones, which was associated with the hypertrophic exostosis due to pyorrhœa. He had an idea that there might possibly have been some infection, and there was some reason to think that it had increased in size. In dealing with the case he was cautious, and said that it could do no possible harm. It was allowed to remain, and had not since increased. He had also examined some of the lower animals in order to see whether there was anything analogous to this condition. The question, on taking up the study of animals, was as to whether he should restrict himself to a large thing like a hump or a breastplate, or take note of a ridge—a heaping-up of bone along the suture line. He decided that it would be better to take note of every ridge. Among animals there was considerable variation between the flat palate and the palate presenting a ridge. Among the higher apes there were very few ridges, and not many among the carnivora, but practically all classes of vertebrate animals showed ridges to a greater or lesser extent. In ruminants and other animals a ridge in the front part affording a protection to the anterior palatine nerves and vessels was the more obvious, while in man it was in the back part that it was chiefly in evidence. The only animal in which he found a well-marked bossing behind the palatine foramen was *Hystrix africa*. He had found the ridge also in the manatee, and, in fact, in practically every class

of animal examined it was discovered, but with no regularity in any one. It appeared to him to be a normal variation of the palate, and whether it was going to show itself merely as a suture line or as a broad plaque they had no indication.

Mr. STANLEY MUMMERY asked whether Mr. Godlee considered that all bony swellings in the middle line of the hard palate were of this nature. Symmetrical exostoses of the lower jaw were not uncommon, and in one of the cases which Mr. Godlee had mentioned the swelling undoubtedly had increased in size, as he (Mr. Mummery) had had this patient under observation for several years. It apparently began to increase in size about four years ago and became flatter, and they saw that a similar alteration had taken place in other cases. Mr. Heath had mentioned the case of a woman who noticed a small, hard bony swelling in her palate when she was quite a child. This increased to a certain size and then remained stationary until she was 40 or 43 years of age, when it began to increase in size rapidly. The patient came to Mr. Heath to ask his advice, and on removing the growth he found that it was undoubtedly cancerous. If this was exostosis, did Mr. Godlee consider that there was any danger of these changes taking place in the case referred to?

Mr. HERN said that in the course of his practice he had come across a few of these cases. They were rare in his experience, although not very rare. During the last twelve or fourteen years he had seen at least six cases. Mr. Godlee possessed the models of some of them. With regard to Mr. Godlee's Case No. 1, in which he (the speaker) was concerned, he was now absolutely sure from his colleague's evidence that the sister who had seen her, respecting whom there was at first some doubt, had a torus of about the same size as the other. He had been in the habit of taking a model in these cases in order to see whether there was any subsequent increase in size. He thought that such models might be useful at any future time if a surgeon required proof as to whether the swelling had or had not diminished. His own experience in watching these cases was that they did not change much. Some time ago when Mr. Godlee asked him whether he had seen any of these cases he was pleased to be able to show him a few models. Since then he had been asking his dental colleagues whether they had any models showing the torus, and one of them told him that he had unfortunately recently destroyed a model which he had possessed for twenty years. This was to be regretted, because it was the model of a patient who was aged 60 when it was taken, and who had recently died at the age of 80. His friend saw the patient shortly before her death, and the torus had not apparently increased in size at all.

Mr. GODLEE said that he was very much interested in what Mr. Butlin had stated about the patient whose torus had apparently grown to a certain extent. He did not find many of the abrupt tori in the skulls he had examined, and it was possible that the size of the swelling depended somewhat upon thickening of the mucous membrane. The bony growth might therefore appear to increase or diminish, whereas the real cause of the changes was

more or less superficial. Dr. Keith had dealt with the anthropological side of the question, and on this he had only to add that the inhabitants of prehistoric Britain had most remarkable palates. Almost all those that had been preserved among the prehistoric skulls at Oxford showed more or less indication of the presence of a torus and were extraordinarily rough. It was very interesting to hear what Mr. Turner had said about his observations among the lower animals. He (Mr. Godlee) had always felt that he ought to have gone further than the examination of the primates, and he thought it probable that further investigations in this direction would reveal some interesting facts. In answer to Mr. Stanley Mummery, he could not pretend to say that every bony swelling in the palate was a torus palatinus, but he felt confident that Mr. Mummery's cases were both genuine examples of it. With regard to ivory exostosis, he had not come across a single case in the examination of between two and three thousand skulls, and he thought it must be very rare. It was also interesting to hear about the small proportion of tori found among lunatics. At the Oxford Museum, where he saw the fairly large collection of sixty skulls, about ten out of the sixty had a torus palatinus. The asylum from which they were taken, however, was a military one, and probably the skulls were those of people of several different races.

Surgical Section.

March 9, 1909.

Mr. J. WARRINGTON HAWARD, President of the Section, in the Chair.

Urachal Cyst simulating Appendicular Abscess: Arrested Development of Genital Tract; with Notes on Recently Reported Cases of Urachal Cysts.

By ALBAN DORAN, F.R.C.S.

OVER ten years ago I read before a meeting of the Royal Medical and Chirurgical Society a communication entitled, "A Case of Cyst of the Urachus with Notes on Urachal and so-called 'Allantoic Cysts.'" Several years passed by, but I came across no further examples of urachal cyst in my own practice until last summer, when I revealed by operation a remarkable and, I must add, undiagnosed example of this form of tumour. It simulated appendicular abscess, and was associated with arrested development of the upper part of the genital tract. I will relate this case and then dwell upon others recently reported by Mériel, Weiser, Binnie, Delore and Cotte, E. D. Ferguson, &c., adding an unpublished report of a cystic sarcoma of the urachus in the practice of my friend Mr. F. S. Eve. I shall discuss almost exclusively pure urachal cysts as distinguished not only from tumours which are not urachal but also from cystic urachal fistula, which, like other forms of urachal fistula, is clinically and surgically quite different from urachal cyst. The consideration of the pure cyst, in itself somewhat complicated, is quite sufficient for a single paper. I will endeavour to explain how much has been added to our knowledge of these cysts since I reported my first case in 1898.

THE CASE.

F. C., aged 17½, single, a dressmaker's apprentice, applied to Dr. Drummond Maxwell at the Out-Patients' Department, Samaritan Free Hospital, on July 16, 1908. She complained of tenderness and swelling in the right iliac fossa, associated with a history of a sudden attack of pain in that region a month previously, and she was admitted into my wards at once. After admission I found that the relations of the swelling to adjacent organs could not well be defined until I examined the patient with the aid of anæsthetics under circumstances presently to be explained.

The patient's mother informed me that the catamenia were established at the age of 14, without pain or constitutional disturbance. The periods were always scanty and attended with very little pain, and the interval was about five weeks. The patient had never suffered from any neurosis before, at, or after puberty.

On June 16, one calendar month before admission, the menstrual flow appeared as usual, but was accompanied by violent pain never experienced before. The pain continued for two days and then abated. The patient at once resumed her work as a dressmaker, but the pain returned two days later and obliged her to take to her bed again. During the whole of the week before admission she was quite incapable of attending to her duties.

Roughly speaking, as regards what could be made out before anæsthesia was employed, there was a fairly well-defined, almost spherical, swelling in the right iliac fossa, slightly movable and tender to touch. There was resonance on percussion over its outer aspect. The lower part of the swelling could be defined on rectal examination. I refrained from making a vaginal exploration until a consultation was held. Then it was found that the vagina was barely two inches deep. A kind of dimple could be felt at the blind extremity towards the right. The tumour did not bulge into the vagina. At the lower limits of the swelling was a tuberosity which lay behind the vagina and in front of the rectum. The temperature and pulse were low. The patient had never been laid up with any severe illness. Before the arrested development of the vagina had been detected, appendicular abscess was suspected, but after the examination hæmatometra or hæmatosalpinx seemed equally probable.

On July 21 the period began, as usual, about five weeks after that

which preceded it. I found that there was no palpable increase of pain or tenderness in the tumour nor any appreciable increase or decrease in size. The show was unusually free. I decided to examine the patient under anæsthesia during the period in order to discover the channel which transmitted the menstrual blood into the vagina, and for other manifest reasons.

Examination under Anæsthesia.

The patient was a fairly healthy but slightly anæmic blonde. Her manner and appearance were perfectly feminine. Though hardly over 5 ft. in height she was well proportioned, broader at the hips than at the shoulders, and free from hair on the face. The breasts were well developed; there was no areola round the nipples. The axillary and pubic hair corresponded to the patient's age. The inguinal canals contained no tender body. The perineum was markedly deep, so that the anterior commissure lay far forward. The labia, clitoris and meatus urinarius were normally developed. There appeared, on the other hand, to be no hymen, nor was there the least trace of carunculæ. The vagina formed a blind pouch about 2 in. deep; the rugæ were prominent. The vaginal pouch was distinctly deeper on the right side, whence dark menstrual blood was seen to issue. On stretching the adjacent mucosa with the fingers a crescentic fold with the concavity towards the left was detected. It covered the aperture whence proceeded the blood. A uterine sound could be passed into this aperture and pushed onwards for 3 in. upwards, backwards and a little to the right, closely following the outer limits of the lower pole of the swelling, as could easily be defined on digital exploration from the rectum (fig. 1).

On bimanual palpation the swelling was found to be a well-circumscribed tumour, firm, oval and as tense as a recent hæmatocele. It could be pushed a little downwards, yet even then its lower pole did not bulge into the vagina, but passed behind it. The tuberosity in the recto-vaginal septum, discovered at the previous examination, lay to the left of the menstruating tract. It felt like a small cervix.

The nature of the case remained obscure. I kept the patient at rest for a week. The period ceased and the tumour remained stationary. There was one sharp attack of local pain on July 28, without any rise of pulse or temperature.

Operation.

On July 29 I operated with the assistance of Dr. V. Monckton, Dr. Belfrage administering ether and chloroform. I made an incision in the middle line. The parietes were unusually vascular. After separating the recti I came across a thick membrane of doubtful character, and lower down I exposed the wall of the bladder which extended for quite 2 in. above the pubes. The membrane was cut through, and about half a pint of a perfectly clear fluid was removed; unfortunately none was preserved. The fluid lay in a cyst behind the recti and anterior to the parietal peritoneum, the membrane through which I had made the incision being the anterior portion of the cyst-wall. The cyst was connected with the bladder by a thick cord $\frac{1}{2}$ in. in length. The upper limits of the cyst lay close below the umbilicus. In exploring the upper end of the tumour I laid open the peritoneal cavity. The omentum adhered to the peritoneum investing the back of the cyst. The intestines seemed healthy; there was no evidence of tuberculous disease, no free fluid, and no intraperitoneal tumour. Below the omentum some coils of ileum adhered to the parietal peritoneum behind the tumour.

I endeavoured to define the relations of the cyst to the genito-urinary tract. A catheter was passed into the bladder, and a few ounces of urine were drawn off. There was no communication between the cavity of the bladder and the cavity of the cyst; the thick cord between the two was clearly a portion of the urachus, and I observed that it ran into and not over the cyst-wall.

As might have been suspected from what could be defined before the operation, the cyst lay to the right of the middle line. On pressing against its wall on the right inferiorly, from the inner side, I detected a fusiform body like a uterine cornu, or a small but entire virgin uterus, lying in the position of the menstruating tract along which a sound had been passed a week before. Above this body thickened tissue could be felt, apparently a small ovary. The tuberos, cervix-like body already mentioned could be plainly defined through the walls of the lowest part of the cyst. When thus explored it was found to be a distinct, fairly movable structure, the left ovary or uterine cornu. On farther palpation through the cyst-wall the pelvic cavity felt quite free from any tumour or deposit. There certainly was no such thing as a collection of retained menstrual blood.

At this stage of the operation it became evident that the swelling,

which disappeared entirely when I opened the cavity full of fluid, was a urachal cyst. That swelling—in other words the cyst—had been the cause of all the patient's recent trouble. As there was no trace of a hæmatometra or hæmatosalpinx I did not feel justified in dissecting in the dark behind the cyst, amidst deformed structures in very uncertain relations to ureters, blood-vessels, &c., merely to make out the extent of arrested development of the uterus and appendages.

It was with the cyst, therefore, alone that I had to deal. I knew of several objections to the draining of a urachal cyst, nor could I dissect away its outer wall, since, as I have just observed, its posterior relations to malformed structures were very uncertain. For these reasons I simply trimmed away as much of the lining membrane as could be safely removed. Then I cautiously passed several fine catgut sutures in the substance of the outer wall and tied them, so that the cyst-cavity was closed in. This outer wall was the muscular sheath of the urachus abnormally thickened, so that the manœuvre just described was easy and nothing was caught up behind the cyst.

I transfixed the segment of the urachus which ran between the lower limits of the cyst and the bladder with a fine linen suture and tied it on both sides. It was then divided between the cyst and the ligature. As will be explained presently, it is fortunate that I transfixed the urachus, instead of tying a single ligature round it as though it were an artery. I kept the portion attached to the cyst for microscopic examination.

Lastly, the sheaths of the recti were united with interrupted fine linen sutures and the integuments closed with interrupted silkworm gut.

After-history.

During convalescence there was no difficulty in micturition, which was voluntary from the first, and no urine leaked through the wound. By August 7 all the silkworm gut sutures were removed; the wound was by then well healed. There was at that date no trace of any swelling in the right iliac region or pelvis. The firm movable body which lay until the operation below the lowest part of the cyst was found, on bimanual palpation, to be connected with the fusiform body to the right of the middle line.

During the summer vacation Dr. Maxwell took charge of the patient in my absence. He reported that up till the day of her discharge at the end of August there was no sign of leakage of urine through the wound nor any show of blood.

On September 12 the patient came to see me at the hospital. Her general condition was good. A slight show of blood had been noted on the previous day, the first indication of a period since the operation. The abdomen was flat and free from tenderness. The cicatrix had completely healed. No trace of any abdominal tumour remained; the right iliac fossa was free; there was no resistant area or gurgling on pressure within its limits. No solid or cystic tumour nor any ill-circumscribed resistant body could be defined in the pelvic cavity. The fusiform body which transmitted menstrual blood and the firm movable body on its left were now definable as one tough, smooth, irregular structure which could be pushed up to the level of the pelvic brim, slipping down immediately the examining finger was withdrawn. The mobility of this structure was very marked. The segment towards the

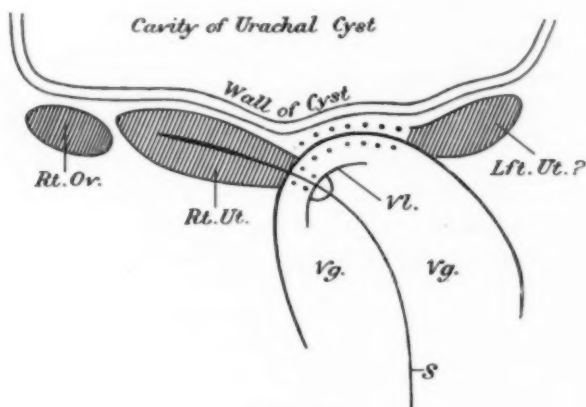


FIG. 1.

Diagram showing the arrested development of the genital tract and the relation of the malformed parts to the cyst of the urachus. *Vg.*, vagina, its blind end rising higher on the right side than on the left; *Vl.*, valvular fold, through which a sound, *S*, passes into *Rt. ut.*, the right cornu; *Ov.*, right ovary; *Lft. ut. (?)*, solid body, probably left cornu, the dotted lines indicate a band, not clearly definable, connecting it with the right cornu.

left was not in the least tender, which would imply that it was the left cornu and not the corresponding ovary. Thus the urachal tumour had disappeared and no hæmatometra had formed, whilst the uterus was represented by a right cornu which communicated with the vagina yet had no cervix, and most probably a left cornu connected with the right by a membranous band (fig. 1).

By the middle of October the patient was in very good health. She had been able to work for nearly four weeks, and a month later she reported herself as quite able to continue at her duties without feeling pain or fatigue.

On December 15, 1908, I once more examined the patient. She was in good health and had menstruated three weeks previously. The local condition was as in September, except that I could distinctly define close to the right cornu the ovary-like body or thickened tissue which I had detected by touch at the operation, and it was tender on pressure like a normal ovary.

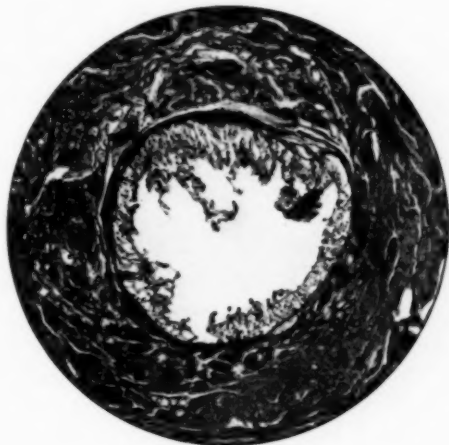


FIG. 2.

Section of the segment of urachus which passed between the bladder and the cyst-wall, as seen under a low power. The canal is quite unobstructed and lined with transitional epithelium; the muscular coat is very thick.

Microscopical Appearances of the Cord between the Cyst and the Bladder.

A section of the cord-like structure which ran on the surface of the parietal peritoneum between the fundus of the bladder and the cyst was made at the College of Surgeons. There could be no doubt that it was a portion of the urachus. Mr. Shattock reported that the canal was quite patulous and lined with perfect transitional epithelium of the bladder type. The lumen was free from catarrhal or other morbid

products. The muscular coat was abnormally thick, but showed no evidence of inflammation or œdema. Its inner portion was mostly made up of circular and its outer portion of longitudinal fibres; but there was some irregularity in the direction of the fibres in both portions. Some subperitoneal fat was intimately connected with the periphery of the urachus. The appended photomicrograph (fig. 2) shows the above-described appearance of the urachus as seen under the microscope.

The urachal canal, according to Wutz, is well developed in young subjects. In this instance it must have been closed at the bladder end, or, if not, there must have been an efficient Wutz's valve, as the contents of the cyst showed no evidence of being fouled by urine.

Having related this case, the second in my experience, I will now turn to the anatomy of the urachus, dwelling on certain peculiarities of importance in respect to the surgery of urachal cysts.

THE URACHUS.

The surgical anatomy of the urachus deserves more attention than it has hitherto received in this country. There is no necessity for us to slight the embryologists; indeed, we can take on trust what A. Keith, Ballantyne, Bryce, Cunéo and Viau, and other authorities teach us, being fairly convinced that the urachus is developed from the allantois. There is likewise no need for us to disparage the labours of Bland-Sutton, Byron Robinson, Freer and others who have demonstrated the pathology and surgery of urachal cysts and urachal fistulæ under their own observation. Let us rather turn our attention to a matter intermediate between embryology and pathology—in other words, let us study the anatomy of the urachus as revealed by special investigation of human subjects other than patients suffering from distinct disease of that interesting embryonic relic. Then, perhaps, we shall be better qualified to understand the pathology, diagnosis and treatment of urachal cysts.

Wutz, so often quoted, stands first among writers whom we should follow as an example. In reporting my first case I noted his observations on the histology of the urachus based on seventy-four post-mortem subjects. He found that the epithelial tubular portion grows steadily up to the twenty-fifth year, the canal becoming wider, which is in accord with the appearances displayed by the microscope in the segment of urachus below the cyst in the present case. Passing over histological details, two

important statements deserve to be noted. In twenty-four subjects in Wutz's series there were distinct cystic dilatations of the urachal canal, which contained pus in two instances where the patients had died from septic affections. Of equal importance is Wutz's statement that the vesical orifice of the urachus is guarded by a transverse valvular fold which, under normal conditions, prevents the passage of urine into the urachal canal. In 1898 I expressed my hopes that these researches would be followed up in this country, but up to the present Wutz has found but few imitators. We want to know more about the valve, for instance. Is it constant? The best recent work after the Wutzian method, as I may call it, has been undertaken by Binnie and Clendening in America. I will quote in full their summary: "Mr. Clendening recently examined for me sixteen adult cadavers and seven foetuses, with the following results: (1) In seven adults and six foetuses the bladder showed a distinct diverticulum from 1 cm. to 2 cm. deep at the fundus where the urachus is attached. (2) In one adult there was a slight projection instead of a diverticulum. (3) In eight adults and one foetus the dome of the bladder was smooth. (4) In none of the cases were there found lacunæ lined with epithelium in the urachus. (5) The average adult urachus was 12 cm. long by 1.5 (0.15?) cm. wide. (6) The urachus was usually adherent to the belly-wall, but in one case (diabetic with frequent retention of urine) it was not close to the parietes, but lay between loops of small intestine. (7) In all the cases the urachus was well supplied with vessels."

Binnie adds: "Early in their development the urinary bladder and the urachus are completely surrounded by peritoneum except on their ventral surface, where a meson (mesocyst)¹ exists. This disposition usually disappears and the urachus becomes extraperitoneal. That the mesocyst sometimes persists was well exemplified in an adult cadaver examined for me, in which the urachus did not lie close to the abdominal wall, but lay between loops of small intestine. The persistence of the meson explains the occurrence of some otherwise puzzling intra-abdominal cysts."

This case was the clinical evidence on which clause 6 in the above summary was based. The meso-urachus question is of high importance in respect to urachal cysts, especially those which have attained a large bulk. Delore and Cotte's researches were published almost simultaneously with the monograph prepared by the two American writers, and, as will

¹ This expression, meaning, of course, mesentery of the bladder, is best discarded, lest it should be taken to signify the mesentery of the urachal cyst.

presently be related, they claim to have detected and removed a true urachal cyst that was intraperitoneal.

The French authorities quote Cunéo and Viau, who have found that in a section of an embryo, 45 mm. in length, the intra-abdominal portion of the allantois and the umbilical arteries are for the greater part completely invested by peritoneum. The disposition persists when the allantois has begun to differentiate itself into urachus above and bladder below. It is, however, transitory, and the urachus becomes, as a rule, extraperitoneal in its entire length.

Professor A. Keith, conservator of the museum of the Royal College of Surgeons, informs me that he has on several occasions detected a very considerable mesentery enfolding the urachus in adult subjects. He adds that under these circumstances the obliterated hypogastric arteries had even deeper and better marked mesenteries. Professor Thane has likewise observed this condition in several adult subjects. In these particular cases there was no evidence, I presume, that the mesentery was acquired, but Professor Keith tells me that he has never chanced to come across a meso-urachus in the new-born infant. Pathological evidence, as I will explain presently, does not lead us to believe that the urachus can acquire a mesentery after foetal life. Mr. Lockwood reminds me that different observers are not agreed as to where a "fold" ends and a "mesentery" begins, but admits that the urachus may be invested in a fold of parietal peritoneum.

Thus, on the testimony and experience of independent embryologists, anatomists and surgeons, we learn that the urachus may possess a mesentery, or at least a fold of peritoneum, representing most probably the persistence of an embryonic condition. Still, we wish to know more about this matter and about Wutz's valve—so important in respect to the question of the precise relation of a urachal cyst to a cystic fistula connected with the bladder. Therefore it were well if some British teacher of anatomy or pathology would follow the good example of Wutz and of Binnie and Clendening. A series of several hundred subjects investigated after Wutz's method could hardly fail to furnish fresh evidence of high value.

THE URACHUS AND DIVERTICULA OF THE FUNDUS OF THE BLADDER.

Although, for reasons given in the introductory remarks at the beginning of this communication, urachal fistula must be dismissed, I may be allowed to say a few words on abnormal prolongations of the

urinary bladder upwards. Garrigues discovered, in a single woman, aged 45, who died after hysterectomy for fibroid, a bladder prolonged nearly to the umbilicus, to which it was connected by a very short urachal tube. Balfour Marshall reports a case where a patent urachus over 1 in. in diameter formed a tubular prolongation of the bladder, and was wounded when an abdominal incision was made for ventrofixation of the uterus, though without bad consequences. I turned attention to these diverticula in 1898,¹ and have noted Binnie's and Clendening's researches above. The surgeon will hardly trouble to distinguish a diverticulum of the bladder from a patent urachus, even if there be any distinction. What concerns him is the fact that such a structure may be wounded during an abdominal section.

TAIT'S PSEUDO-ALLANTOIC CYSTS.

In 1898 I showed how the long series of cases reported by Lawson Tait had been misinterpreted by that great surgeon when he ranked them as urachal cysts, excepting one published by himself and Teichelmann in the *Lancet* twenty years ago. Several writers who have apparently never read my criticisms have recently expressed precisely the same views. Mériel, Binnie, and Delore and Cotte all maintain, as I did, that Tait's series were instances of encysted peritonitis, probably tuberculous, where, as I pointed out, communication between the bladder and peritoneal cavity is not infrequent. I may add that Mr. Tait himself, in a letter about my monograph in 1898, informed me that he had detected mature hair-follicles in sections of the cyst-wall in one case. This would imply that the cyst was a universally adherent ovarian dermoid, or a dermoid tumour derived from some other organ, but urachal it could hardly be.

There were no after-histories to these cases, and no verification of the relations of the cysts at necropsies. Hoffmann's case, where the cyst contained fifty litres of fluid when opened at the post mortem, was shown by Wutz to be an example of chronic hæmorrhagic peritonitis. The original report was so defective that an absurd error about the sex of the patient was included. Yet Delore and Cotte agree with Hoffmann, though rejecting Tait; and in articles on urachal cysts by living writers Tait's series and Hoffmann's equally spurious case are persistently included as though they really represented urachal disease. The error

¹ Loc. cit., p. 307.

seems, like King Charles the Second, a most unconscionable time dying. Let them be henceforth relegated to the archives of chronic peritonitis—in other words, to their proper place.

CASES OF CYSTS OF THE URACHUS RECENTLY REPORTED.

Two years ago W. R. Weiser published tables of nearly ninety reported cases of cysts of the urachus. These tables are indispensable for the study of their subject, but they show, as their author admits, that we cannot as yet draw from the literature of urachal cysts any sound conclusions such as may be inferred from published series of the ligature of big arteries for aneurysm, or Wertheim's operation, where at least there was always the aneurysm or the cancerous uterus to begin with. Weiser includes Tait's and Hoffmann's spurious cases and a considerable number of examples of cystic fistula, two being in his own practice.

Weiser's second case seems an authentic instance of a pure urachal cyst. It bore some relation to my own, as it occurred in a young subject (a girl aged 11); it gave rise to an acute attack of abdominal pain, headache, vomiting and fever, and was markedly unsymmetrical. There was impaired resonance between the umbilicus and pubes from the left loin to about 2 in. to the right of the median line. When the abdominal incision was made the parietal peritoneum was found investing the posterior aspect of a large cyst, which was connected inferiorly with a duct running into the bladder, and patulous, let it be noted, to within $\frac{3}{8}$ in. of the vesical cavity. The cyst almost filled the left side of the abdomen below the level of the umbilicus. It contained pus, and had ruptured at one point into the peritoneal cavity. As much of the sac as could be dissected out without tearing through the abdominal wall was taken away, the cavity left behind was washed out and drained with iodoform gauze, and the patient recovered. Let it be noted that before the operation tuberculous peritonitis was suspected.

Whilst Weiser's¹ own case is well reported, as I have stated above,

¹ Weiser has overlooked a case of cystic fistula reported by Unterberger. A woman had a bad fall during the first month of pregnancy. Acute backward displacement of the uterus occurred, the bladder became distended, and at length urine passed entirely through the umbilicus. Rupture of an adherent ovarian cyst was suspected, but a catheter passed into the umbilicus met another introduced through the urethra. The uterus righted itself, and delivery occurred at the fifth month. Unterberger gives a good summary of cases of urachal fistula and cystic fistula.

no safe inferences can be drawn from his tables, so judiciously prepared that, among other things, the defective character of many of the reports by other writers is to be seen at a glance. Let us trust that the next writer who takes the trouble to tabulate all recorded cases of urachal dilatation will carefully group under separate headings: (1) fistulæ; (2) primary cystic fistulæ communicating from the first with the bladder or opening at the umbilicus; (3) pure urachal cysts, the subject of this communication; and (4) secondary cystic fistulæ, developed from pure cysts which have acquired communications with the bladder or umbilicus. Surgically and clinically the fourth, as well as the second, must be carefully distinguished from the third. Hence I will pass over all statistics and will now proceed to the consideration of cases of pure urachal cyst published during the last ten years, but not included in Weiser's tables. That writer, I must add, omitted Bryant's two cases which were appended to my monograph in 1898. The first was a pure cyst, in a woman, and simulated an ovarian tumour; the second was a cystic fistula, primary or secondary, and in a male patient.

The recent cases which I will now dwell on were under the observation of surgeons who, like myself, have enjoyed the advantage of studying the experience of others, and were thus the better able to avoid the fallacies which beset the original observer. It happens that these recent cases form a group fairly typical of every variety of pure urachal cyst, sessile on the bladder or separate, extraperitoneal or provided with a mesentery, incipient, moderately developed or large—a most instructive series, in fact. We need not dwell on cystic dilatations detected in foetal bodies, but as surgeons we must not disregard certain records of the accidental detection of small urachal cysts in the course of operations.

SMALL CYSTS DETECTED AT OPERATIONS.

Morestin, a few years ago, operated upon a woman aged 24, removing a suppurating Fallopian tube. When making the abdominal incision he brought to light two cysts too small to be detected by palpation. One lay above the other in juxtaposition but independent, and they occupied the middle line behind the recti and in front of the peritoneum. The urachus could be seen running from the fundus of the bladder into the lower cyst and from the upper cyst to the umbilicus. The cysts did not adhere to the peritoneum behind them. They were tense, smooth, globular and transparent. One was opened, and found to contain a limpid colourless fluid. The outer wall consisted of connective tissue;

the inner was lined with pavement epithelium. Let it be noted that there was no trace of a meso-urachus, nor in either of the cysts which I have described, which were both of moderate size, was the tumour invested with peritoneum except posteriorly. This fact would lead us to believe, as was mentioned above, that a urachal cyst does not make for itself a mesentery as it develops. When a urachal cyst is intraperitoneal the urachus most probably had a mesentery before the cyst existed.

Again, we see that Morestin operated for pyosalpinx, a suppurative condition which was localized. When that condition is more diffused an incipient urachal cyst may be involved, as in two instances recorded by Wutz, to which I have already referred. Hence it is always possible that an abscess in the middle line below the umbilicus may have developed in the urachus. Lastly, Morestin's cysts probably represented an incipient bilocular urachal cyst like that which I described in 1898.

Mériel, of Toulouse, laid open a cyst of the urachus when performing a cystotomy for retention of urine. Its walls were thin, but distinctly thicker towards the bladder,¹ into the fundus of which it was inserted. The cyst had three walls, the outer of connective and fibrous tissue, the middle muscular and the inner thin and smooth.

Lastly, the experience of Opitz is interesting to all who undertake abdominal sections. When performing what he calls a *Relaparotomie*—in other words, when repairing an incisional hernia—he exposed a small cystic body which at first sight appeared to be a displaced and adherent vermiform appendix. On closer examination there could be little doubt that it was a urachal cyst. If so, it would seem that a segment of the urachus where the canal happened to be unobliterated was involved in cicatricial tissue at its upper and lower limits, the canal subsequently undergoing dilatation owing to a collection of fluid and broken-down epithelium in its lumen. The main interest of this case is, however, as in Balfour Marshall's experience already mentioned, the manner in which the vagaries of the urachus and fundus of the bladder may puzzle the operator. Whilst the little cyst in this instance simulated a vermiform appendix the big cyst in my own case gave rise to symptoms indicating appendicular abscess.

CYSTIC TUMOURS REMOVED BY OPERATION.

In 1899 E. D. Ferguson operated on a man aged 47; his original report is very carefully written. The tumour rose to 2 in. above

¹ *Vessie* is misprinted *veine* in the original report.

the umbilicus and extended laterally to the iliac spines. Its surface was flat, resistant on pressure, and felt as though in the abdominal wall, yet malignant disease of the omentum was suspected. The chief symptoms were hypogastric pain and frequent desire to make water. It proved to be a cystic tumour containing over two quarts of a watery fluid, which unfortunately was not examined; it was intimately connected with the wall of the bladder and extended deeply into the pelvis, where the peritoneum lay behind it. Ferguson dissected away the whole lining membrane of the cyst, excepting at the umbilicus, where he found digital processes penetrating the tissues of the abdominal wall. "That portion of the posterior part of the cyst which could be placed in a fold and allow easy approximation of the peritoneum was stitched together and excised." The upper part of the abdominal incision, close to the umbilicus, was drained with gauze, the remaining portion carefully sutured. Six months after the operation the abdominal cicatrix was found to be perfectly sound.

Delore and Cotte's intraperitoneal urachal cyst, to which I have already referred, is reported in their instructive monograph on big cysts of the urachus. The patient was a girl, aged 20, very sickly, and believed to be the subject of tuberculous peritonitis of the ascitic type. There was uniform distension with distinct fluctuation. A large cyst was exposed when the parietal peritoneum was excised. It descended into the pelvic cavity. It contained blood-stained fluid and fibrinous masses, and when it was emptied and drawn through the abdominal incision Delore found that it was connected below with the fundus of the bladder by a short cord which formed an entirely extraperitoneal pedicle and was clearly the lowest portion of the urachus. When the cord was divided the cyst was free of all connexions with the patient's body. The broad ligament and other peritoneal folds near the cyst were normal. The patient made a speedy recovery.

The outer wall of the cyst was the muscular coat of the urachus; the inner wall had been greatly altered and deprived of its epithelium by inflammatory changes. The authors describe minutely how they found on careful dissection that the cyst was invested over the whole of its extent by the peritoneum, and that at the level of the attachment of the cord connecting it with the bladder this fold of serous membrane ceased. A tough cord ran from the pedicle over the anterior surface of the cyst, and was lost in the tissues of the tumour and abdominal wall at the level of the umbilicus. In its middle portion this cord, like the cyst, was completely intraperitoneal.

There can be little doubt that Delore and Cotte's tumour was a cyst

developed in a urachus which possessed a mesentery, an exceptional condition observed, however, as above mentioned, by more than one writer in adult subjects where the urachus was otherwise normal. The authors are probably correct in their theory that it represented the persistence of a foetal condition. That, on the other hand, a meso-urachus can be acquired is very doubtful, for reasons given above in reference to Morestin and Mériel's incipient urachal cysts in adults.

The last in this series of recent cases has hitherto been unpublished. Mr. F. S. Eve has presented to the Museum of the Royal College of Surgeons a unique specimen of cystic sarcoma of the urachus, and has kindly supplied me with the following notes.

A man aged 38 was admitted into the London Hospital with a swelling in the hypogastrium noticed for several weeks and associated with pain after micturition. A cystic tumour filled the lower part of the abdomen, especially to the right, where it extended towards the loin. It did not dip into the pelvis. On puncture dark blood came away; a few days later a rigor occurred, with vomiting and a rise of temperature to 104° F. Mr. Eve then operated, exposing a large cystic tumour; the parietal peritoneum was reflected over its anterior and superior surface. Five pints of dark bloody material were removed. The cyst adhered to the omentum, which bore engorged veins, and to an inch and a half of small intestine which was infiltrated where adherent. The adherent portion of the wall of the gut was excised and the wound closed with sutures.

The lower part of the cyst was intimately connected with the bladder, the serous coat of which organ was reflected on to its surface. This peritoneal covering was divided and the cyst carefully dissected away from the bladder. During this process the bladder was opened, for the vesical wall at this point was so thin that the cavities of the cyst and the bladder were only separated by the vesical mucous membrane covered by a few muscular fibres. The opening was sutured, but not without great difficulty owing to the thinness of its walls at this point. The sutures were further protected by gauze packing. A gauze drain was passed into the pelvis and a catheter retained for a while in the bladder. Neither flatus nor fæces could be made to pass after the operation, and the patient died on the fourth day. There was no general peritonitis, but the pelvic peritoneum had become inflamed at the point where the gauze had been applied.

Mr. Eve examined the specimen and found that it was a large allantoic cyst separated from the posterior superior surface of the bladder by nothing except a very much thinned mucous membrane. Their

cavities, however, did not communicate. The inner wall of the cyst was lined at certain points with very vascular polypoid masses which proved on microscopical examination to be sarcomatous. The most unusual feature of this cyst was its malignancy, but its peritoneal relations were of greater importance in respect to the subject of this communication.

Aveling and Bland-Sutton have already reported a case of multilocular myosarcoma of the sheath of the urachus, but it did not involve the urachal canal and was quite unconnected with the bladder. The specimen No. 417 B in the Pathological Series of the Museum of the Royal College of Surgeons was supposed, when first examined, to have developed in the urachus, but Mr. Targett considered that it was a myosarcoma which originated in the connective tissue surrounding the bladder.

Mr. Eve's cystic sarcoma seemed to possess a partial peritoneal investment as in Delore and Cotte's case, but it was sessile on the bladder, whilst in the latter case the cyst was separated from the bladder by a segment of urachus. In the cases of extraperitoneal urachal cyst above noted, the tumour was sessile on the bladder in E. D. Ferguson's case, and so also was the bilocular cyst which I reported in 1898. On the other hand, in the case which is the subject of this communication, the cyst and the bladder were separated by a segment of urachus. These opposite relations of cyst to bladder were demonstrated respectively in the incipient tumours discovered by Morestin and Mériel.

The malformation of the genitals, a unique feature in my case, deserves a little consideration. After discussing it I will dwell once more on the extreme asymmetry of the cyst, which, together with the inflammatory complications in its neighbourhood, simulated appendicular abscess. In conclusion I will comment on the surgery of pure urachal cysts.

RELATION OF THE URACHAL CYSTS TO THE ARRESTED DEVELOPMENT OF THE GENITAL TRACT.

In my case it was clear that there was not a cystic urachal fistula, but a pure urachal cyst which was separated by a short cord, the lowest part of the urachus, from the bladder. There was no evidence of malformation of the bladder itself; its fundus and anterior portion as well as the urethra were normal, nor was there any sign of abnormality in the ureters sufficient to interfere with their functions. The structures making up the vulva were also well developed, and the lower part of the vagina was present, nor had either genital gland strayed into the inguinal canal.

On the other hand, the upper part of the genital tract showed marked arrest of development. The short canal which transmitted menstrual blood into the right side of the blind end of the vagina represented the upper part of the vagina, the cervix, and more or less of a right uterine cornu with its endometrium. The firm, movable body above and behind the blind end of the vagina was most probably the left cornu connected with the right by a thin band, as is not rarely seen in cases of uterus unicornis in parous women. These relations were not difficult to define after the patient had recovered from the operation, and no cyst remained to interfere with bimanual palpation.

I admit that the body which felt like the left cornu might have been the left ovary, in which case the cornu would more probably be suppressed altogether. I made out during the operation an ovary-like structure close to the right cornu. The presence or absence of the Fallopian tubes and the disposition of the pelvic peritoneum remain mysterious, though there was no trace of a hæmatosalpinx or a hæmatometra. Although there were no external signs of hermaphroditism, I cannot say positively that both genital glands must have been ovaries. Dr. A. Broca recently operated for double inguinal hernia on a subject who was a female according to all external appearances.¹ The vagina, clitoris, and hymen appeared normal. Yet both genital glands were true testicles. Each occupied a hernial sac.

Some pathological change early in foetal life must have caused the two abnormal conditions. Professor A. Keith, in his recent museum demonstrations at the College of Surgeons, explained how there was every reason to believe that disease of adjacent maternal structures, such as endometritis, prejudicially affects the normal evolution of foetal elements derived from the allantois, and the same may be said of the gradual conversion of Müller's duct into Fallopian tubes, uterus, and vagina. For the cystic condition of the urachus and the arrested development of the uterus in my case, there must have been a common cause. The urachal cyst presumably represented in a very mitigated form the arrest of development known when extreme as ectopia vesicæ.²

¹ Compare the case recently reported by Arnolds in the *Monatsschr. f. Geb. u. Gyn.*, Berl., 1908, xxviii, p. 463, where an operation was performed on a married man aged 59, subject to diabetes, for the removal of a tumour of the left testis. The "tumour" proved to be a uterus bicornis with well-developed Fallopian tubes and a pair of testes each with an epididymis, but neither had a vas deferens. See also Cranwell, "Les Hernies Inguinales de l'Utérus," *Revue de Gyn. et de Chir. abdom.*, Paris, 1908, xii, p. 777.

² Epispadias is the rule in ectopia vesicæ in males. A. Keith finds that hypospadias is not associated with this condition. In Bryant's second case of urachal cyst, or rather cystic fistula (*vide supra*), there was slight hypospadias.

In this distressing variety of malformation, and also in complete ectopia of the abdominal viscera, non-union of Müller's ducts has repeatedly been observed (E. Chill, author). According to A. Keith the uterus is double in one out of six specimens of ectopia vesicæ in London museums, whilst in seven specimens of ectopia in females combined with an open yolk sac Müller's ducts are unfused in all.¹

The faulty condition of the genital tract in my case was of little surgical interest, as it was not of a kind such as could be remedied by the knife or by some plastic procedure. It was the condition of the urachus which demanded operative interference.

ASYMMETRY OF URACHAL CYSTS.

In the present case the cyst lay in the right iliac fossa, the middle line forming its limits to the left. Some intestine was adherent to the cyst on the right, so that there was resonance on percussion, and there was likewise local pain and tenderness. Thus an asymmetrical urachal cyst may simulate appendicular abscess. Arguing without clinical evidence, we might conclude that a urachal cyst would develop symmetrically. This conclusion, however, is belied by experience. In the remarks on recent reports given above it was noted that in Weiser's case the cyst filled the left side of the abdomen, whilst Eve's cystic sarcoma lay mainly to the left. The cyst removed by Douglas, of Nashville, in 1897, was also asymmetrical, distending the abdomen chiefly to the right.

This asymmetry becomes less surprising when we remember that it often exists from the beginning. Delore and Cotte, speaking of minute urachal cysts as described by pathologists, observe that these dilatations sometimes involve the entire lumen and circumference at the point of the urachus where they develop, but that as a rule they affect only a portion and form true diverticula which may become completely isolated from the canal of the urachus. Thus the pathology of this asymmetry is very clear and its clinical importance quite evident.

ASSOCIATION OF URACHAL CYSTS WITH PERITONITIS.

The symptoms of peritonitis in this case where there was a conspicuous swelling in the right iliac region indicated appendicular abscess. Peritonitis had developed around an asymmetrical cyst of the urachus.

¹ "Malformations of the Hind End of the Body," *Brit. Med. Journ.*, ii, 1908, pp. 1736, 1804 and 1857.

The first attack of pain occurred at a menstrual period, so that when the atresia was detected, I naturally suspected retained menses. But the swelling did not increase during the next monthly period when the patient was under my care, and I found at the operation that it was not a hæmatometra or a hæmatosalpinx. We must therefore turn back to the local peritonitis. The patient in this case was evidently exposed to some unfavourable influence which caused peritonitis around the cyst.

In my first case, reported in 1898, the patient, aged 59, had been subject for nine months to pain in the lower part of the abdomen; the cyst was then detected, and symptoms of peritonitis came on periodically until two months later, when I operated. I find, on referring to my original case-book, that the menopause had become complete over nine years before—that is, when the patient was about fifty; in fact, there is no reason to believe that the catamenia had anything to do with either case. In both I found omental and intestinal adhesions to the parietal peritoneum lining the back of the cyst. Other writers, Delore and Cotte, Weiser, &c., have reported similar complications. Indeed, peritonitis seems to be the rule when a urachal cyst has reached proportions sufficient to give rise to symptoms leading to its detection. These symptoms appear very irregularly, which is one reason why *encysted dropsies* and *tuberculous peritonitis* are so apt to be confounded with urachal cysts. The error of mistaking the former for the latter, into which Hoffmann and Tait fell, has, as I have been at some pains to explain, greatly impaired the value of statistical records of urachal tumours.

AGE.

In the present case the patient was 17 years of age, in Weiser's she was only 11, and in Delore and Cotte's 20. Urachal cysts are relatively frequent in young girls; and as they are likewise subject to tuberculous peritonitis and to inflammation of the vermiform appendix, we can see how easily errors of diagnosis may occur.

SURGICAL TREATMENT OF PURE URACHAL CYST.

Any communication between the interior of a urachal cyst and the cavity of the bladder, or the surface of the body at the umbilicus, greatly increases the difficulties and dangers of operative measures. It is partly on that account that I have confined my observations almost entirely to pure urachal cysts. Whenever it seems fairly clear that a cyst of this kind can be extirpated without the dangers of dissecting in the dark

amidst uncertain relations posteriorly, the radical operation should be undertaken. This, however—itself not free from risk, even under the most favourable circumstances—is not always possible, as shown by the reports of many surgeons; whilst in the present case the posterior wall, for special reasons already given, could not have been safely separated from the abnormal structures in close relation to it. We must remember that in urachal fistula the insertion of the ureters into the bladder may be abnormally high (Mikulicz), and we are dealing with a malformation closely related to fistula. The surgeon must in many cases be content with partial measures which, as I have explained, were successful in Weiser's, E. D. Ferguson's, and my own two operations. As much as possible of the inner lining membrane must be dissected away; in the present case this was readily effected, but in my 1898 case it was impracticable, the membrane having been destroyed by degenerative changes. Partly on that account, and partly because the tumour was bilocular, I found drainage necessary. A sinus developed but closed within six months, nor did it ever open again. The patient died of cancer of the pylorus and pancreas six years later. In the present case there was no indication for drainage. The firm and fairly thick main wall of the cyst, fibromuscular or purely fibrous, is very favourable for treatment by whip-stitching or any similar plastic procedure suitable according to circumstances.

During the operation, complete or partial, the surgeon must always carefully ascertain the relations of the cyst to the bladder. If the cyst be sessile, it may be impossible to remove it without damage to the walls of the bladder, which will therefore require repair by suture. When, on the other hand, the cyst is separated from the bladder by a segment of urachus, it must be remembered that the segment may have an open canal communicating with the bladder. The microscope showed that in the present case it was open (fig. 2), although there was no proof that it ended unobstructed in the cavity of the bladder. Lest a urinary fistula should develop, as appears to have happened in a case described by Ill many years ago, that writer treats the divided segment as though it were a vermiform appendix, turning in its cut edges and then applying a Lembert suture. In the present case I transfixed the segment and tied it as though it were a pedicle of an ovarian cyst. In any case it should not be tied without transfixion, as though it were an artery. The loop might easily loosen or come away and the canal would then be patulous. We know that this may happen when the stumps of both Fallopian tubes have been ligatured in a double ovariectomy, as pregnancy has followed,

which means that the canal of one tubal stump must have transmitted an ovum from the relics of one ovary. Should the ligature come away from the urachal stump, a urinary fistula of a very bad type would develop if Wutz's valve were forced open by over-distension of the bladder. The risk of such a complication under these circumstances and the fact that a pure urachal cyst in males is occasionally converted into a secondary cystic fistula full of urine reminds the surgeon that, as I have already remarked, we ought to be more sure about Wutz's vesico-urachal valve or whatever it may be that really protects the vesical orifice of the urachus.

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DISCUSSION.

The PRESIDENT (Mr. Haward) said he was sure the Fellows would wish to accord their best thanks to Mr. Doran for his important paper. Any such contribution regarding difficult cases of abdominal surgery must be of great value, and it would be interesting to hear the experiences of other surgeons concerning urachal cysts. It was to be hoped also that some further investigations might be made by anatomists with regard to the anatomy of the urachus in the adult, as suggested by the author. An interesting point was the frequency with which urachal cysts were found on one side of the middle line, and it was important for surgeons to remember that fact in connexion with their diagnosis. It was somewhat remarkable that so many of those cysts suppurated.

Mr. F. EVE said he thought Mr. Doran's paper was of interest and importance, not only pathologically, but also to the surgeon. He happened recently to come upon Weiser's paper in the *Annals of Surgery*, to which Mr. Doran had referred, and was surprised to find that that authority had collected the records of eighty-nine cases of urachal cysts. Some of them, as Mr. Doran had suggested, might be apocryphal, but if three-fourths were really cases of urachal cysts, that was a considerable number. One point which struck him in glancing through the list was the large number of cases which suppurated. He also noticed the difficulties which attended the operation in many cases; in a considerable proportion the operation was an incomplete one, consisting in opening the cyst, removing part of its walls, and draining. He understood that was the way Mr. Doran treated his case, and he therefore asked him whether he had any subsequent difficulty with regard to the fistula. Mr. Doran had referred to a case which came under his (Mr. Eve's) care, and of which the specimen was on the table. The author had given all the important details regarding the clinical history. The most important point anatomically in regard to his own case was the fact that the greater portion of the anterior surface of the cyst was lined with peritoneum. Mr. Doran suggested the explanation that it was lined with peritoneum because the urachus had a mesentery, and as the cyst enlarged it expanded the mesentery and became covered by it. He (Mr. Eve) thought an equally satisfactory explanation was to suppose that in the formation of such a large cyst, which projected upwards and to a large extent backwards, it invaginated the peritoneum before it, and (without having a mesentery) acquired on its anterior and upper part a covering of peritoneum by a process of involution. This explanation occurred to him at the time, but he did not then know there was ever such a thing as a meso-urachus.

Possibly he might have been thought to be over-bold in attempting to remove the cyst. Mr. Doran had already stated that the man, immediately before the operation, while in the hospital, had a severe rigor, and the temperature went up to 104° F. He therefore expected to find a suppurating cyst, but found a cyst filled with blood, which had evidently become septic. It was evidently of the utmost importance that something should be done. On opening the cyst it was also found to be definitely malignant, and that fact determined him to attempt its removal. It was separated very carefully, but he thought it would have been impossible for anyone to have separated it from the bladder without opening that viscus, for at the point where it was connected with the apex of the bladder there was practically nothing more than mucous membrane between it and the vesical cavity. It was surprising to him that surgeons, in opening the abdomen, did not more often come upon remnants of urachus at some point between the umbilicus and the apex of the bladder. He could not remember on any occasion to have met with anything which looked like a remnant of the urachus. He had only met with one other example of urachal cyst, and that was a specimen which was brought to the College of Surgeons many years ago from Clare Market. It was now preserved at the College. The cyst, as large as an orange, was attached to the apex of the bladder of an ox. At the apex of the bladder

near the attachment of the cyst was a small convoluted tube, which was a continuation of the urachus to the umbilicus.

Mr. W. G. SPENCER observed that Mr. Doran said suppuration was rather frequent, and he wished to ask him whether he had formed any opinion why it was so. Was there a preliminary extravasation of blood, and where did the infection probably come from? Supposing the patient had quite healthy urine, would the infection come from the intestines? One knew that the infection came from the bladder in old men's stricture.

Mr. ALBAN DORAN, in reply, said that in regard to Weiser's series it was difficult to get percentages. There were certainly a number of spurious cases in the list. The same authority showed that suppuration was common. Some germs might travel along the canal of the segment of the urachus below the cyst, where perhaps urine would be unable to pass, or a few minims of urine might get into the cyst occasionally and become septic there. But that was not the rule. He thought it probable, as Mr. Spencer suggested, that there might be infection from adherent gut. It was common for the parietal peritoneum at the back of such cysts to be adherent to the intestine. That happened in his first patient markedly, and to some extent also in the second. In some instances there had been communications with tuberculous intestine, and that explained why there was also suppuration. With regard to drainage, in his first case, where the cyst was bilocular, which he did not recognize until the end of the operation, he was obliged to drain, because he had opened a cavity lined with diseased mucosa. No urine came away, and the patient was watched by Mr. Blamey, of Penryn, and she died of another malady long afterwards, the wound having remained strong. In the second case Mr. Doran did as Ferguson in America had done—that is, he tore out or dissected away as much of the lining membrane of the urachal cyst as possible, and whip-stitched the raw surface of the outer coat of the cyst, which was the muscular coat of the normal ureter much hypertrophied. With regard to the perforation of the peritoneum in Weiser's case, he agreed with Mr. Eve. Although some foreign observers said that the urachus never made itself a fold of peritoneum secondarily, Mr. Doran believed that Mr. Eve was correct when he said that in his (Mr. Eve's) case the cyst made itself a mesentery by involution, pushing back the parietal peritoneum until the folds of the serous membrane met to some extent in front of it. In some of those cases where there was a relatively small cyst close to the bladder, it was best to resect the bladder for about a quarter of an inch or more all round the attachment, otherwise a bad fistula might develop. Mr. Doran had had a large experience of abdominal sections, and he had repeatedly seen dilatations of the urachus such as were described by Wutz and Delore and Cotte, and he had observed that the muscular sheath of the urachus seemed to be thickened in cases of myoma. If Fellows of the Society, whenever they operated for uterine fibroids, would watch as they made the abdominal incision, they would almost certainly find the urachal cord very thick, as in the microscopic specimen which he exhibited ten years ago, which was figured in the *Transactions of the Medico-Chirurgical Society*.

It was a hypertrophied sheath with no trace of a canal, taken from a patient on whom he operated for uterine fibroid. The muscular fibres were perfect. But besides mere dilatations, he found that even incipient urachal cysts were extremely rare, and the only cases he had observed in the Samaritan Free Hospital since he joined the staff in 1877 were that which he himself recorded in 1898, and that which formed the subject of the present communication.

The Application of Continuous Suction in Surgery.

By H. T. HERRING, B.S.

THE use of continuous suction as a means of removing blood during operation, and of subsequently draining wounds and withdrawing secretions, has recently received some attention. Dr. R. H. Woods, of Dublin, published an interesting paper in the *British Medical Journal* on May 20, 1905, "On the Treatment of Purulent Cavities," and a second entitled, "On the Saliva Ejector as a Surgical Instrument," in the *Journal of Laryngology*, February 2, 1906. In the latter he dealt with the subject as applied to nose and throat operations, and I wish now to endorse what he and others have said, and to advocate a more extended trial of this agent in general surgery.

I have systematically employed continuous suction, as opposed to intermittent aspiration, for many years, and there can be no question of the benefits conferred on patients, especially after the operation of cystotomy, both as regard their comfort and their general well-being; for they are kept thereby quite dry, and thus relieved of the infliction of frequent dressings, which are imperative under ordinary circumstances. Dryness and the relief thus afforded undoubtedly enable wounds to heal more rapidly, and tend to a lower mortality from post-operative causes.

The chief obstacle which has up to the present prevented the more general employment of continuous suction is, I think, the want of some portable apparatus which can be used in any place, and which will easily maintain a continuous negative pressure without much attention. Neither Cathcart's pump nor G. W. Richardson's modification entirely fulfil these requirements.

My first endeavour to overcome this difficulty was with a modified form of Plunkett's well-known saliva ejector, fitted with a Royles' patent tap-union, by which it could be attached to any water supply (fig. 1).

This has proved most successful in many cases. I have used it for more than seven years, and others have tested its capabilities. It works well as a rule if the water pressure is good, though its action is not very rapid and rather liable to variation. But the great objection to it is that the motive power—*i.e.*, the water supply by which it is worked—is very frequently inconveniently situated with regard to the patient's room, thus rendering a long length of tubing necessary to bring the suction to the place where it is wanted. With the appliance which I have here that objection is obviated (fig. 2). It consists of a circulating pump, which will mechanically extract air, and a small electric motor to drive it, both being enclosed in a portable box. As almost every house and room has now an electric installation the motive power

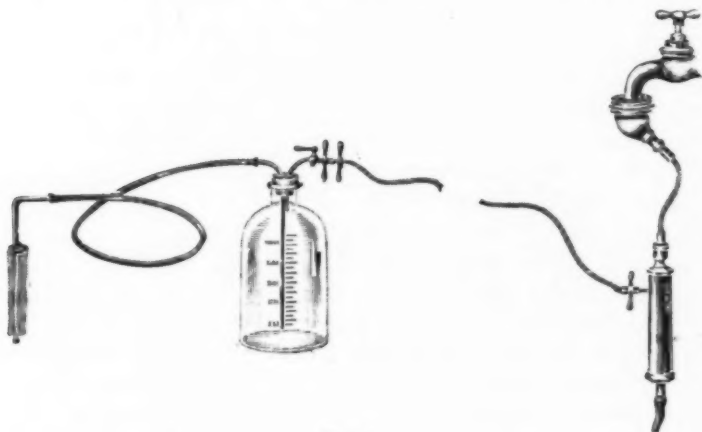


FIG. 1.

A saliva ejector attached to a water-tap, with receiver to catch extracted fluids and a gauze suction-tube for insertion into a wound or cavity.

exists everywhere, and always close at hand. Connexion between the motor and the nearest lamp-holder has only to be established and the pump is ready to work. This apparatus is exceedingly efficient, extracting 10 to 12 cubic feet of air per hour, and will produce a high negative pressure (over 29.5 in. of mercury). It will run for days, requiring only to be lubricated occasionally. The weight of the whole apparatus is about 26 lb., and the cost of running it does not exceed a shilling a day, reckoning the B.T. electric unit at sixpence.

Perhaps I may now be allowed to mention one or two other points

which have to be provided for in order that suction may be successfully applied in surgery :—

(1) To prevent the blocking of the suction tube in the wound, either by the tissues themselves or by blood-clot, mucus or pus.

(2) To provide for the drainage and removal of fluids collected in cavities, or dependent parts not actually reached by the suction tube itself.

The easiest method of draining is to insert the end of the suction tube into one of considerably larger diameter, placed in the wound in the ordinary way. The latter acts as a caisson, or well, to collect the fluid, and from it the fluid is sucked as soon as it reaches the level of the aspirating tube. This plan has a disadvantage, for although it

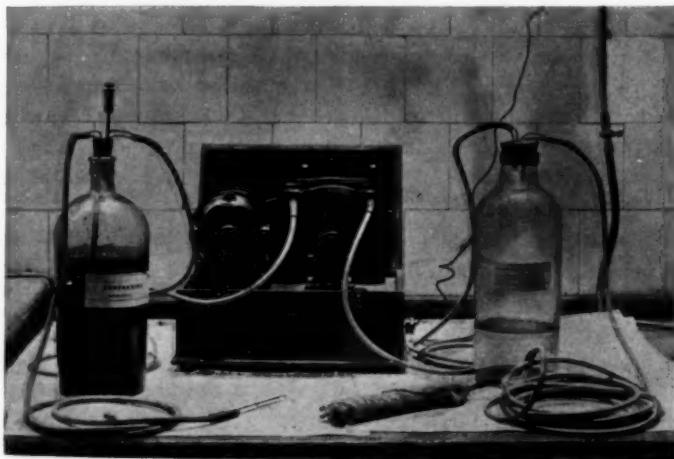


FIG. 2.

Electrically driven air-pump, fitted with receiver and suction-tube on the right and irrigation-bottle on the left.

prevents overflow and succeeds in keeping the cystotomy patient perfectly dry, yet it does not drain the cavity, and always allows a pool of fluid to remain below the level of the suction tube. If, however, the caisson is replaced by a porous or absorbent one, which I effect by tightly wrapping several layers of lint or gauze round the lower three inches of the suction tube, the result is much better. The fluid is first soaked up into the material and is then gradually extracted from it

and carried away by aspiration. Of course, the roll of material must extend beyond the lower end of the tube and be long enough to touch the floor of the cavity, and at the same time project above the surface of the skin; for otherwise atmospheric pressure will be unable to act on the cavity and a negative pressure will be established there, when extraction of fluid will at once cease.

Another application of this principle is simply to embed the end of the aspirating tube deeply among the dressings. By either means extraction continues as long as any part of the absorbent dressing touches fluid; so that secretions, &c., percolating into a cavity are at once absorbed by the material, and then extracted and carried away to the receiver by the suction action. The receiver of which I have just spoken is a large bottle interposed between the pump and the suction tube. It catches and retains all the fluids withdrawn, and prevents the blocking of the apparatus.

This pump may also be used to irrigate a wound with any solution desired. One end of a small tube is attached to the outlet of the pump, the other to the short arm of a Woolf's bottle filled with the irrigating solution. A second tube—that which is to convey the fluid to the wound—is connected with the arm extending to the bottom of the bottle. The action then of the pump is to force air into the bottle and create a positive pressure, which, in its turn, drives the solution along the irrigating tube to the dressings in the wound. If a small and continuous supply of solution is wanted the irrigating nozzle must be fine, and the pressure regulated by adjusting the escape valve connected with the Woolf's bottle. Thus a wound may be drained and irrigated at the same time without discomfort to the patient.

Suction has been employed in draining the chest, the kidneys, the gall-bladder, the throat and nose, and I think with this more convenient and powerful apparatus a further trial should be given, not only in these cases but also in all where it is important to remove secretions and discharges.

With regard to the removal of large quantities of water, for instance, when a cavity is being flushed during an operation the calibre of the suction tube must be considerably increased and provided with a tap which can be turned off, so that the receiver can be partially exhausted before being used. In this way very large quantities of blood and fluid can be rapidly withdrawn in a short space of time.

Messrs. Maw and Sons, Messrs. Allen and Hanbury, and other instrument makers, have kindly consented to lend the apparatus to anyone who may care to give it a trial.

Surgical Section.

May 11, 1909.

MR. J. WARRINGTON HAWARD, President of the Section, in the Chair.

On Auto-Inoculation and Reinfection of Syphilis.

By J. HUTCHINSON, F.R.C.S.

(1) AUTO-INOCULATION OF PRIMARY CHANCRES.

It is often stated as a character of primary syphilitic sores that they cannot be inoculated on the same individual. Although clinical and experimental evidence agrees in pointing to a rapid spread of the syphilitic virus throughout the system, it would indeed be strange were it true that auto-inoculation is impossible after the sore has existed but a few days. It is a fairly frequent occurrence to meet with two indurated chancres situated on parts of the body which are not directly continuous but which touch each other from time to time: for example, the penis and scrotum, the two labia majora and minora in women, the upper and lower lip on the face, &c. These are, in all probability, examples of auto-inoculation, but they are open to the explanation of simultaneous infection with the spirochæte in two places.

For instance, a patient of mine came under care five weeks after two large indurated chancres had been first noticed; one was placed on the outer skin of the penis, the other on the scrotal skin exactly where the parts were liable to touch. His belief was that only a short interval, if any, had elapsed between the appearance of the two sores. Mr. Waren Tay has reported the case of a man with a copious secondary eruption and an indurated chancre on the prepuce; on the inner side of the thigh, where the parts touched, were three dusky indurated sores. He stated that about two weeks had elapsed between the appearance of the penile and thigh chancres.

Such evidence takes us but a short way, and were no other forthcoming we might be ready to accept the dogmatic assertion of one of the most recent writers on the subject. Colonel Lambkin¹ says of the primary chancre "it is not readily auto-inoculable, and *only so during the first ten days of its existence.*" He proved the possibility of auto-inoculation from the penis on soldiers within ten days of the existence of the primary sore. "After ten days I found it impossible to reproduce an identical sore or anything like it; as a rule a small inflammatory pustule was the only result. From later experiments made by myself I find that mercurialization of the patient renders auto-inoculation almost impossible, even during the first ten days."

The following cases, by no means all that have come under my observation, prove that nature may provide better experiments than those of our own devising:—

Case I.—A single woman, aged 25, came under care with a typical indurated chancre of the lower lip, accompanied by the usual sub-maxillary bubo. A full three weeks later another, but smaller, indurated chancre developed on the upper lip in an exactly corresponding position.

Case II.—A gentleman, after exposure to risk, had an indurated chancre on the penis with inguinal bubo. Nearly a month later (*i.e.*, between three and four weeks) a sore developed on the last phalanx of his right thumb. This gradually assumed the circular raised form of an infecting chancre, and was attended by an axillary bubo. A syphilitic eruption on the trunk followed quickly, with condylomata about the anus and sores on the tongue. It cannot be doubted that he inoculated the thumb from the penile chancre, and the length of the interval was absolutely certain. I believe he was taking mercury during the weeks referred to.

Case III.—A patient at the Lock Hospital had a large indurated chancre on the end of his penis in October. In December (fully two months later) after he had been foolish enough to continue poulticing the organ, a second indurated chancre developed at the root of his penis, several inches away from the first sore. There was a large bubo, and a papular eruption developed. He had no proper treatment, and in May following—*i.e.*, no less than eight to nine months after the first inoculation—both chancres were still large and indurated. The sore which appeared the latest was the more active and angry-looking of the two. The remarkable persistence of the two chancres is noteworthy and very unusual.

¹ Power and Murphy, "System of Syphilis," 1908, i, p. 190.

Case IV—This is the most remarkable instance of auto-inoculation I have ever seen. It proves that even when general secondary symptoms have appeared a typical fresh chancre with bubo may develop. The facts are undoubted, as I was seeing the patient every week. A man came to the Lock Hospital with an unusually large, hard chancre of the prepuce, which had existed some little time and was attended with a characteristic bubo. He was put on internal mercurial treatment, but this was not commenced in time to prevent a secondary rash and sore-throat. He attended regularly for two months, and then he drew my attention to a "sore finger," which he attributed to a scratch received in his daily occupation, that of a wire-worker. It should be mentioned that he had dressed the sore of the penis every day. On his right index-finger was a commencing sore which looked most like a chancre; in the corresponding axilla the glands were already enlarged. In the next week or two this second sore became larger, circular and indurated. Long, hard, lymphatic cords developed up the arm, and the axillary bubo became very marked. With continuance of the mercurial treatment all the symptoms ultimately subsided.

These cases of auto-inoculation are interesting in several respects. The nearer to the date of first infection that the second inoculation occurs, the more likely is it to succeed in producing a true chancre with glandular bubo. In this, I think, surgeons will agree with Finger and Landsteiner, who say: "Reinoculation is successful in proportion to its proximity, in point of time, to the primary inoculation. If general infection is not yet complete, a typical chancre can be produced, but from the time when constitutional symptoms appear it becomes more difficult to succeed. During the secondary period the result has some resemblance to a secondary papule . . ."

Cases II, III, and IV that I have narrated prove that the second sore even from a late auto-inoculation may, however, have the usual features of a Hunterian chancre and in no way resemble a secondary papule, as stated by Finger and Landsteiner. Cases II and IV are noteworthy from the fact that the chancres were so far apart, on the genital region and finger and thumb respectively. The length of time that elapsed in Case IV, a full eight weeks between the appearance of the chancres, makes it possibly unique. It is stated by many workers at the pathology of syphilis that the *Spirochæta pallida* is found with the greatest difficulty, if at all, in well-indurated chancres. Dr. M'Intosh, who possesses special skill and experience in this matter, tells me that he made an exhaustive examination of sections of seven indurated

chancres without being able to demonstrate the spirochæte in any of the cases. In the early stage of the sore it can often be found. The local use of mercurial preparations, the intramuscular injection of mercury, the administration of it by mouth, all seem to hasten the disappearance of the spirochæte from the primary sore. In Case IV it is obvious that although the patient was under mercury for some weeks, and the indurated sore, which was of quite exceptional size, was improving, yet the virus was transmitted to the finger.

There is an obvious conflict of evidence, as to the persistence of the virus in a primary sore, between pathological and clinical observers. But the clinical evidence as to the danger of infection from an indurated chancre, even when its secretion is practically *nil* or when the sore has almost disappeared, is so strong that it will take much more to discredit it than these observations as to the behaviour of the spirochæte, interesting though they are.

One recent writer¹ has stated his belief that in every case of inoculation of the syphilitic virus there is an abrasion of the epithelial surface, whether skin or mucous membrane, which allows the spirochæte to enter. It is a point of considerable practical importance, but one hard to solve, or rather it is hard to prove to those who picture a pre-existing fissure at the site of every chancre that none was present. Here again we may have to disregard experimental evidence derived from inoculation of chimpanzees. I can only express my firm belief in the frequent entrance of the syphilitic virus through unabraded or normal skin and mucous membrane, and I think the cases of auto-inoculation help to confirm this belief.

(II) REINFECTION WITH SYPHILIS.

Recorded evidence of two attacks of syphilis in the same individual is by no means abundant, and what there is often will not bear scrutiny, as essential points are wanting in many of the case-narratives. Where, for example, the observer narrates the occurrence of primary and secondary symptoms, followed after some years by the development of a fresh sore which was called a chancre and was treated with mercury, and claims this as a proof of reinfection, it is plain that he proves nothing whatever. The recurrent pseudo-indurated chancre, to which my father and Professor Fournier first drew attention, is easily mistaken for a second infection. I have seen many cases of this recurrent induration

¹ Dr. F. W. Andrewes; Power and Murphy, "System of Syphilis," 1908, i. p. 114.

at varying periods from the original attack, and have come to regard it as a tertiary infiltration of tissues at or close to the site of the primary chancre. My experience has been that glandular enlargement—*i.e.*, any form of bubo—hardly ever accompanies it, and that the induration yields more readily to iodides than to mercury. It is unnecessary to say more about a condition that must be familiar to most surgeons, but which has been a fertile source of mistakes in diagnosis.

In testing the evidence as to a true second infection with the syphilitic virus we must be careful in our definitions and stringent as to the facts recorded. Dr. F. W. Andrewes has put the matter well:¹ "By a true second attack should be meant the occurrence, twice in a patient's life, of a primary sore followed by secondary symptoms, and if this definition be accepted the phenomenon must be admitted to be of extreme rarity. As Neisser points out, it means not only that the patient has been completely cured of his first attack, but that his tissues have so returned to the normal as to react to the syphilitic virus like those of an intact person, and, it may be added further, that he has lost any immunity which may have been conferred by his first attack."

I doubt if Dr. Andrewes is quite correct in stating that "the phenomenon must be admitted to be of extreme rarity." At any rate, it is worth while to bring forward such evidence as exists on the question, and it is of interest to note that private and not hospital practice furnishes nearly all the cases which are conclusive. It is, moreover, only those who have to treat cases of syphilis from its outset who will obtain any evidence of true reinfection; indeed, most physicians seem to ignore its possibility. Thus Dr. F. W. Mott² made publicly this year the emphatic statement, "we know . . . also that one attack of syphilis confers immunity during the rest of the individual's life." How far this is from the truth will, I think, be conclusively shown by the following series of cases. The question turns on two considerations: first, the really efficient treatment of syphilis; and, secondly, after such treatment has been gone through that the same individual should be exposed to contagion a second time. With these postulates granted there need be nothing unexpected or surprising in true second attacks of syphilis. The subject is especially worthy of study as helping us to define "really efficient mercurial treatment," as will be discussed later on.

¹ Power and Murphy, "System of Syphilis," 1908, i, p. 165.

² "Morison Lecture" at Royal College of Physicians; reported in *Brit. Med. Journ.*, 1909, i, p. 528.

Case I: Interval between the Two Infections, seven years; Second Attack the more severe and protracted, especially the Eruption on Skin and Mucous Membrane.—Mr. H. had been originally treated by me for stricture of the urethra. He was gouty, and had eczema from time to time, as well as herpes on the penis. After exposure to contagion on November 10, 1894, he came to me with two hard chancres of the penis, one at the preputial furrow, the other on the outside skin. The glands in the right groin were swollen, and a slight secondary eruption, &c., developed. He took hydrarg. cum cretâ fairly steadily for about three years, and, with the exception of recurrence of a cutaneous syphilide in 1896, remained free from symptoms. In September, 1901—i.e., seven years after the first attack had commenced and four years after leaving off mercurial treatment—he was exposed to risk of contagion, and subsequently saw a doctor for a sore on the penis and a bubo. The treatment given was partly mercurial and partly iodide, but he did not improve, and came to me on November 25, 1901. There was then an oval scar on the skin of the penis; the right inguinal glands were “shotty” and enlarged; there was a copious eruption on the trunk, face, neck and thighs, consisting of large macules and papules; the cervical glands were, perhaps, slightly enlarged. In December both tonsils showed typical ulcers and filmy patches. In spite of mercurial treatment (chiefly hydrarg. cum cretâ, but sometimes mercury and iodide mixture) he had relapses of throat ulceration and mucous patches on the lips. He was an inveterate smoker. It was found to be much more difficult to administer specifics during this second attack, but with intervals mercury in some form or other was given for the greater part of three years. I believe since 1904 he has remained well, but there is no question that the second attack of syphilis in this case was both more severe and more resistant than on the first occasion. It was, however, impossible to induce him to give up alcohol during the course of treatment.

Case II: Interval less than three years; Both Attacks mild and amenable to Treatment.—A gentleman was exposed to contagion about March 24, 1900. He noticed a sore on April 15. When I saw it on May 4 it was a typical indurated chancre on the inner surface of prepuce, secreting very slightly, with double indolent inguinal bubo. The nature of infection being thus beyond doubt, I put him on 4 gr. to 6 gr. of grey powder daily. He was very regular as to mercurial treatment, but had bouts of indulgence in alcohol, which somewhat interfered with its efficiency. Relapses of superficial glossitis along the edges of the tongue and the occurrence of a typical syphilitic ulcer of one tonsil

proved that the secondary symptoms had not wholly been prevented by the early adoption of mercury. In addition, he had follicular tonsillitis from time to time during the eighteen months he was under supervision and treatment. On the whole the symptoms were slight, and from the end of 1901 I believe he was quite free until November 12, 1902—i.e., two and a half years after the contraction of the first attack of syphilis, and one year after leaving off mercurial treatment. On this date he came to me again with a typical raised indurated chancre of the penis. This had followed a fresh contagion. There was an indurated lymphatic cord at the root of the penis and an indolent bubo in the left groin; a fine erythema arranged somewhat in ring form was present on the thighs. He took a mercurial course steadily for this second attack, and developed no further symptoms.

Case III: Interval twelve years; Second Attack the more severe.—Mr. R. in 1887 was treated by Prof. R. W. Taylor, of New York, for what was described as a mild attack of primary and secondary syphilis. The history as to the symptoms was quite conclusive. The treatment, with mercury and iodide, was kept up for two years. He remained free from reminders. In 1899—twelve years after the onset of the first attack and ten after leaving off treatment—he was exposed to risk, and developed a chancre of the frænum, which lasted several weeks and was attended by a characteristic bubo. Subsequently he had pustular or ecthymatous sores on the scalp and rather deep ulcers of the upper lip. I prescribed mercury and iodide, but as the patient left for America I was unable to follow up the case for long.

Case IV: Interval only eighteen months, the shortest on record; Secondary Symptoms in both yielding well to Treatment.—A young man, in September, 1892, came to the Lock Hospital with a large excavated chancre of the glans penis, with indurated indolent bubo, and subsequently ulcers in each tonsil. The induration at the site of the chancre lasted for five months. He was treated under Mr. Arthur Ward and myself for seventeen months with mercury. In February, 1894, only eighteen months after the first infection, he came to me with a fresh indurated circular sore on the penis, which was followed in due time by a copious eruption on the trunk, by an indolent bubo, and by general enlargement of the cervical glands. These symptoms yielded satisfactorily to mercury. I would draw special attention to this case, which was exceptional in the shortness of the interval (in fact the treatment of the first attack was only just completed when the patient became reinfectd). The symptoms in both were strikingly complete, and this is the only hospital case of the kind that I have notes of.

Case V : Second Attack at end of nearly three years ; Secondary Symptoms only slight ; Recurrent Induration a year later.—Mr. D., in February, 1902, consulted my father and one of the surgeons on the staff of Guy's Hospital for what was considered by both to be undoubted primary and secondary syphilis. He took mercury for eighteen months, until September, 1903. In December, 1904—*i.e.*, nearly three years after the onset of the first attack—he was exposed to contagion, and I saw him on January 16, 1905. He then had four circular sores on the scrotum, one of which was slightly indurated. I could at first detect no enlarged glands, and was disposed to doubt the chancres being infecting ones. However, typical condylomata ani followed, and I then prescribed hydrarg. cum cretâ, which he took steadily for a year. The condylomata soon disappeared, and no further syphilitic symptoms ever developed. In 1906, after further contagion, he came with a slightly raised and indurated sore on the under surface of the prepuce ; it cleared off under local treatment with calomel, &c., and I regarded it as an instance of the recurrent pseudo-indurated sore.

Case VI : Two Attacks at interval of seven years ; both very amenable to Mercury.—Mr. H. First attack at age of 23 ; chancre followed by eruption and later by gummata. He took mercury, which, in his words, "acted like a charm." Second attack at age of 30—*i.e.*, seven years after the first. A chancre was followed by an eruption, and sores on his throat and lips. He took mercury for a year and was apparently cured. Eleven years later, at the age of 41, I treated him for syphilitic orchitis of one testis which proved very obstinate.

Case VII : Eight years interval between the Two Infections.—A patient was treated at the age of 22 years by Sir T. B. Crosby for primary chancres followed by deep ulcers of the throat. He was under mercurial treatment for about two years, and had no reminders later. When aged 30, eight years having elapsed, he came to me with a large cartilaginous chancre almost at the site of the first one, in the retro-preputial fold. There were bullety glands in both groins. I withheld mercury for a week or two and a fine papular secondary eruption duly appeared on his trunk, completing the diagnosis. Under internal administration of mercury the case did excellently.

All the cases narrated conform to the stipulation laid down at the outset : that the true syphilitic nature of the second infection should be proved by the development of secondary symptoms. Were one to include cases in which mercurial treatment has been adopted as soon as the surgeon was satisfied in his own mind that the second chancre

was syphilitic, it would be easy to extend the list. The seven conclusive examples I have adduced suggest the following deductions:—

(1) Efficient treatment by a continuous course of mercury for one or two years is the surest way of rendering a patient susceptible to second infection. With this proviso he may contract syphilis again within two or three years of the onset of the first attack (Cases II, IV and V). The treatment of the first attack, in all the seven cases, was by a steady mercurial course lasting from one to three years.

(2) The interval between two attacks of syphilis may be so short a time as eighteen months (Case IV)—*i.e.*, the patient may no sooner have finished his course of treatment than fresh exposure may produce a complete fresh attack. The average interval has been in my experience six years.

(3) The second attack may be slighter or more severe than the first: nothing positive can be laid down on this point. If the symptoms on the first occasion have readily yielded to mercury, they will probably do so on the second.

(4) There is no reason why the same patient should not go through even three attacks of syphilis, provided the first two have been well treated. My father has recorded one definite example of this and another much more doubtful one.¹

I wish especially to draw attention to the paper on "Second Attacks of Syphilis" which has been just quoted from. Unfortunately, it was published in a work circulated only amongst private subscribers, and the remarkable collection of facts my father brought forward may have escaped notice. He has reported (*loc. cit.*, pp. 17, 107, 255) no fewer than fifty-six cases under his own observation. But of these he only claims thirty-two as appearing beyond dispute. I have gone carefully through all the case-narratives, and believe that eighteen only out of the fifty-six would be admitted as true second attacks of syphilis, under the qualification rightly laid down by Dr. Andrewes—*i.e.*, the occurrence of primary and secondary symptoms on both occasions. These undoubted cases are Nos. 1, 3, 7, 8, 10, 12, 16, 17, 24, 26, 31, 32, 38, 46, 47 in his list, with three others not tabulated.

The conclusions just laid down would apply equally well to the total number—twenty-five cases—as to the smaller one—seven in all—reported in this paper. It is of special interest to find that three or four of my father's cases support the contention that true reinfection may

¹ "Hutchinson's Archives of Surgery," vi, pp. 113 and 114.

occur within two years of the first. This would hardly be credited without such positive evidence.

Much has been said and written of late, by the advocates of mercurial injection and courses of inunction, against the treatment of syphilis by long-continued administration of mercury by the mouth. Many Continental surgeons speak of it only in terms of disparagement, and some of their English followers are quite as dogmatic, with quite as little cause. Major Lambkin¹ "has long since abandoned internal medication for syphilis." Sir Felix Semon stated his experience "that the treatment of syphilis by small doses of mercury given by the mouth was not protective against secondary and tertiary syphilis." Dr. Lieven, of Aix-la-Chapelle, went so far as to say that "the introduction of hypodermic injections in England meant a blessing to the thousands suffering from syphilis." This is hardly the place to enter into a discussion of such a large and controversial subject. It may, however, be pointed out that the system of mercurial injections for syphilis is no new thing in England, as Dr. Lieven apparently supposed; that it has been extensively tried here for thirty years or more, and that most of the advocates of prolonged internal treatment prefer it as being in their experience the most efficient and undoubtedly the least inconvenient method, the safest and the most free from awkward complications.

Dr. Lieven, who praises the system of intramuscular injections in the curious terms just quoted, admits that "in our country [*i.e.*, Germany], owing to the many disadvantages, if not dangers, associated with injections, the majority of the profession prefer treatment by inunction." It should be noted that his routine course of inunctions covers a full two years, and that many other Continental authorities insist on intermittent periods of treatment for three, four, or even more years. If injections and inunction are greatly superior to continuous administration by the mouth, why should it be necessary to carry them on for a considerably longer period than suffices with a thorough course of the latter method? This is a simple question to which I can at present see no answer. As a matter of personal experience, many of the worst and most obstinate cases of tertiary syphilis I have seen have been in patients treated in the early stage either by intermittent courses of injections, or by inunction at Aix-la-Chapelle or elsewhere.

The chief interest of second attacks of syphilis lies perhaps in the light it throws on the efficiency of treatment. In practically every

¹ *Proc. Roy. Soc. Med.*, ii, No. 4, Laryng. Sect., p. 66.

one of the twenty-five undoubted cases now brought forward mercury had been continuously administered by the mouth for a long period, usually from one to two years. In several of these cases only a short interval had elapsed before the patient again contracted the disease, and his symptoms were exactly the same as if the virus had attacked virgin soil. That such a series of cases can be adduced from the practice of my father and myself is at least a fair argument in favour of the continuous method of treatment.

It should be remembered that the late Professor Ricord, with his great experience of syphilis, stated categorically that he had never met with an instance of two attacks of syphilis in any of his patients. "La Science ne possède pas encore un seul exemple probant de réinfection syphilitique. Ce n'est pas que je nie la possibilité d'une répétition du chancre induré, au contraire j'y crois et j'y crois fermement, quoique l'expérience clinique m'en ait, jusqu'ici, refusé des preuves." May it not have been that Ricord's method of treatment, by interrupted courses of mercurial inunction, was responsible for this failure of evidence as to the possibility of reinfection?

If it be admitted that two attacks of syphilis may be acquired, and that such an occurrence is not very rare, we should expect to find abundant evidence of the subjects of inherited syphilis acquiring the disease in later life. The longer the interval, the greater should be the chance of reinfection. The inherited syphilitic has been infected before birth; surely in twenty or thirty years all protection or immunity should have been lost. Personally I have had seven years' experience at the Lock Hospital (where there is an immense amount of acquired syphilis), about ten years at Moorfields Hospital (which is, perhaps, the finest field for the study of the inherited disease in young adults), and over twenty at the London Hospital (where there is abundance of both acquired and inherited syphilis). I have been constantly on the look-out for cases in which the double infection could be clearly proved, yet doubt if I have seen more than three or four in all. The following case is conclusive; its subject was a man, aged 28, who attended the out-patient department of the London Hospital with a hard chancre of the penile furrow, double inguinal bubo, and secondary eruption of annular and crescentic form which was especially marked on the scrotum. Thus his acquired syphilis was undoubted, nor was the evidence of inherited taint less marked. The bridge of his nose was much sunken, he had chronic ozæna, symmetrical deafness (from internal ear trouble), whilst both corneæ showed nebulae from old interstitial keratitis. In the *Archives*

of *Surgery*, vol. v, p. 75, there is a solitary example of the same kind recorded, but in this case the evidence of inherited syphilis rested on the patient's history rather than any objective signs.

No doubt the apparent rarity of such cases—acquired syphilis in the subjects of inherited taint—is to be explained by the fact that so few adults show conspicuous traces of the latter. At the same time it is probable that severe inherited syphilis with late symptoms (those occurring about puberty, for instance) does confer a much longer immunity than an ordinary acquired attack for which the treatment has been prompt and effective. It is interesting to note that the *Spirochæta pallida* can be demonstrated with greater ease and abundance in the late lesions of inherited than those of acquired syphilis.

To the two cases at present adduced I could add a few more from the literature of the subject. The late Sir Alfred Cooper and Mr. Edward Cotterell published in 1895 a paper on "Syphilitic Reinfection," consisting almost entirely of cases recorded by other observers in England, on the Continent, and in America. The only original case adduced in this paper was one, under Mr. Cotterell, of "a woman, aged 20, who has well-marked syphilitic incisor teeth and evidence of old keratitis, yet she is going through an ordinary attack of constitutional syphilis, which began with a well-marked indurated chancre." Similar cases were described by Professors Bœck and Lang, Dr. Dowse, E. Arning, and my father, but the details are very scanty and some of the evidence doubtful. The same criticism must be made of many of the case-records collected by Sir A. Cooper and Mr. Cotterell of two attacks of acquired syphilis in the same individual. A large proportion wholly fail to carry conviction to the reader's mind, though some are conclusive.

DISCUSSION.

The PRESIDENT (Mr. Haward) said that he was sure that the Fellows would wish to accord their best thanks to the author for the interesting paper, which added many important facts to their previous knowledge and showed that evidence of care in its preparation which they were accustomed to associate with the name of Hutchinson. He was particularly interested in the case of auto-inoculation while the secondary eruption was still apparent. Another interesting point upon which it would be useful to hear the opinion of other surgeons was how far the occurrence of a second attack of syphilis depended upon the efficiency of the preceding treatment. Anything which enabled one to

judge of the relative efficacy of the different methods of treatment of syphilis was of great interest. He was glad to hear Mr. Hutchinson's testimony to the utility and efficiency of treatment by the mouth.

Dr. McCULLOCH said that in the cases of chancre which the author had described, the element of secondary and tertiary infections from the abraded surface seemed to have been overlooked. He referred to the probability of streptococcic infection, in which there was lymphatic glandular involvement. The latter he was inclined to regard as a mechanism of defence. Those glands being generators of leucocytes, the glandular involvement was really an effort to produce cells leading up to the production of anti-bodies. And where there were anti-bodies evoked for more than one infection—spirochæte infection and streptococcic and perhaps staphylococcic infection at the site of the abrasion—the processes were much more complex, with resulting breaking down of the defence and abscess formation. In those cases the acquirement and duration of immunity would not be expected to be so perfect as where the infection was simply pure spirochæte infection. In his opinion the more toxic the micro-organism concerned, the more active was the lymphatic gland involvement, and, in the light of present clinical research, the terms "hard" and "soft" chancre did not adequately express the essential nature of the disease. The failure of a true spirochæte inoculation in the "soft" variety might be due to its admixture *in situ* with more resistant micro-organisms which teemed in the genital passages.

Mr. HUTCHINSON, in reply, said he fully admitted that there were different poisons in those sores, and in the most interesting of the cases in his paper there were, no doubt, other micro-organisms present. But what he relied on was that a sore developed on that man's finger which might have been a septic sore at first, and that then it gradually assumed the typical raised, rounded, projected characters of a chancre. And those who had seen many cases of digital chancre would admit that while at times they had no characteristic features, at others they were typical, and in the case referred to he had no doubt as to the diagnosis. Moreover, the hard lymphatic cords up the arm—not the lymphangitis which faded away after a short time, but lymphatic cords persisting, with no sign of abscess formation—were very suspicious. Finally, the patient had a large indolent bubo in the axilla.

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Jejunal and Gastrojejunal Ulcer following Gastrojejunostomy, with Notes of Two Cases, in one of which Gastric Analyses were made before and after Operation for Jejunal Ulcer, with an Abstract of Sixty-one other Cases and Observations thereon.¹

By HERBERT J. PATERSON, M.B., F.R.C.S.

NOTWITHSTANDING all that has been written in recent years on the subject of gastrojejunostomy, there remains at least one problem in connexion therewith in need of further investigation and elucidation. The fear of the possible occurrence of a jejunal ulcer still casts a shadow—faint, it is true—over the otherwise admirable results which follow this operation when performed in appropriate cases. It is of the utmost importance that all instances of this condition should be published, in order that we may have all the available material from which to draw conclusions as to its ætiology, as to the best methods of preventing it, and of treatment should it occur.

The notes of the following case which has been under my care are of interest, as it is, so far as I can gather, the first case in which an attempt has been made to obtain an accurate analysis of the gastric contents, and of the fluid escaping from a jejunal fistula, before and after operation for ulcer of the jejunum.

ANTERIOR GASTROJEJUNOSTOMY FOR PYLORIC STENOSIS; PERFORATION OF A JEJUNAL ULCER FIVE YEARS LATER; RESECTION OF AFFECTED PORTION OF JEJUNUM, AND ANTERIOR GASTROJEJUNOSTOMY "EN—Y" (ROUX'S OPERATION).

H. I., married, aged 47, was admitted into the London Temperance Hospital on August 21, 1902, under the care of my colleague, Dr. Soltau Fenwick, suffering from pain after food, vomiting, and loss of flesh.

History: Patient has always been healthy until two years ago, when she began to suffer from pain after food, which has gradually increased

¹ The expenses of the investigations referred to in the course of the paper were defrayed by a grant from the Science Committee of the British Medical Association.

in severity. Since then she has lost 3 st. in weight. For one year she has suffered from vomiting, which has been worse during the past month. She states that the vomiting occurs two or three times a week, large quantities of partially digested food being brought up.

Condition on admission: Patient is very much wasted and weak; weighs 6 st. 5 lb. 4 oz. (normal weight about $9\frac{1}{2}$ st.). Abdomen much distended in upper part. Stomach apparently greatly dilated, lower border reaching a hand's breadth below the umbilicus; constant visible peristalsis; marked succussion splash. At the level of the navel, close to outer border of right rectus muscle, is a hard, movable lump about the size of a walnut. Marked free hydrochloric-acid reaction in stomach contents after test breakfast, and also in contents of fasting stomach. No lactic acid present.

August 27: Patient has had a good deal of pain since admission, but no vomiting. The stomach has been washed out daily, large quantities of residual food being drawn off.

Dr. Fenwick thought that the dilatation of the stomach was due to cicatricial stenosis of the pylorus or duodenum, and asked me to see the patient with a view to operation. The patient declined to have anything done, and left the hospital on August 28.

Patient returned to the hospital on September 6, looking a good deal worse and stating that she had been vomiting constantly. She had lost over 4 lb. in weight, and now readily consented to operation. On September 8 I opened the abdomen in the middle line. The stomach was large and much hypertrophied. In the pyloric region was a densely hard mass, about the size of a walnut, and situated mainly on the posterior wall. There were no adhesions and the lump was freely movable. Anterior gastrojejunostomy was performed by means of a single row of Halsted's mattress sutures, eighteen sutures in all being employed. The opening between the stomach and jejunum was made 2 in. long. The pylorus was explored from within, and a hard, almost complete stricture was found. The patient vomited once before leaving the theatre, not afterwards. She made an uninterrupted recovery—highest temperature after operation 98.8° F., highest pulse rate 72—and left the hospital three weeks later.

This was one of my earlier gastrojejunostomies, and I must admit that, at the operation, I thought the pyloric lump to be malignant, and I proposed to reopen the abdomen, remove a portion of the lump for microscopical examination, and if, as I anticipated would be the case, the lump were malignant, perform partial gastrectomy. Clearly, I was

wrong in my opinion, but fortunately the patient felt so well that she declined to have any further operation.

I saw the patient again two years later, in August, 1904. She told me that since leaving the hospital she had never had a day's illness or pain of any kind, and that she was able to eat ordinary food. She looked a different being, and weighed 9 st. 4 lb., a gain in weight of over 3 st. The abdominal wound was soundly healed, and no sign of the pyloric lump could be felt.

On August 13, 1907, the patient was readmitted into the hospital. She stated that ever since the operation in 1902 she had been in excellent health. Two years ago she began to have some pain after food, but the pain was not severe and did not cause her much inconvenience. During the past nine months the pain has increased. Nevertheless she has continued to eat all kinds of food—cheese, pickles, tinned foods, &c. For three months the patient has noticed a lump forming in the abdominal wall above the umbilicus. On August 13, 1907, the lump burst and much yellowish fluid escaped. She was seen by Dr. Edmunds, who at once sent her up to the hospital.

Condition on admission: Patient looks well, and is now exceedingly stout and weighs over 12 st., a gain of 6 st. since she was last in hospital. Midway between the umbilicus and the ensiform cartilage, immediately to the left of the middle line, is an aperture in the abdominal wall with shelving indurated edges, and from which is escaping a turbid yellowish fluid apparently containing bile. Around the aperture there is considerable induration, and the skin is much thickened for some distance around. Diagnosis: Perforated jejunal ulcer.

As the patient was so well nourished, I thought that it would be advisable to postpone operation with a view to getting the skin into a more healthy condition, and also to reducing the gastric acidity and rendering the intestinal contents sterile by keeping her on an exclusively milk diet. This period of waiting was utilized to make a series of examinations of the gastric contents and of the fluid escaping from the fistula.

August 20: Test breakfast (tea and dry toast); one hour later stomach contents drawn off, 110 c.c., and analysed:—

Total acidity	85.0
„ chlorides	0.433
Free hydrochloric acid	0.072
Protein	„	0.208
Mineral	„	0.153

During the digestion of the test breakfast, and for half an hour afterwards, the fluid escaping from the fistula was collected and analysed, with the following result:—

Total chlorides	0.405
Free hydrochloric acid	0.044
Protein	"	0.175
Mineral	"	0.186

On August 27, after the patient had been on a milk diet for a week, another test meal was given. Analysis:—

Total acidity	80.0
Gmelin's reaction	absent			
Total chlorides	0.361
Free hydrochloric acid	0.018
Protein	"	0.187
Mineral	"	0.146

At the same time 60 c.c. of fluid were collected from the fistula. Analysis:—

Gmelin's reaction	present			
Total chlorides	0.372
Free hydrochloric acid	0.004
Protein	"	0.092
Mineral	"	0.276

On September 3, 70 c.c. of fluid were collected from the fistula. Analysis:—

Total acidity	60.0
Gmelin's reaction	present			
Total chlorides	0.401
Free hydrochloric acid	0.004
Protein	"	0.109
Mineral	"	0.292

Analysis of fluid collected from the fistula on September 4:—

Total acidity	61.0
Gmelin's reaction	present			
Total chlorides	0.324
Free hydrochloric acid	0.006
Protein	"	0.113
Mineral	"	0.304

September 5, 1907: Chloroform; an oval incision was made through the skin around the fistula and carried downwards through the fasciæ and muscles. The peritoneum was cut through in the middle line well below the fistula, and a finger introduced into the peritoneal cavity. The jejunum was found adherent to the abdominal wall for some

distance on either side of the fistula. The fistula was plugged with gauze, and the peritoneum was carefully cut through clear of the area adherent to the jejunum. It was then apparent that the fistula led directly into the efferent limb of the jejunum, 1 in. below the site of the anastomosis with the stomach. Six inches of jejunum, including the perforated ulcer and skin attached to it, were resected. After separating the jejunum from the stomach, the opening in the stomach measured 1 in. by $\frac{1}{2}$ in. This opening was enlarged and the distal end of the jejunum was implanted into it, and the proximal end of the jejunum was implanted into the side of the jejunum 4 in. below the anastomosis with the stomach. Each anastomosis was performed by means of two



A portion of the jejunum resected in author's case, showing jejunal ulcer.

continuous sutures. Great difficulty was experienced in suturing the abdominal wound, as after removal of the sinus and dense hard tissue around it there was a considerable interval between the edges of the wound. It is interesting to note that all trace of the pyloric lump felt at the first operation had disappeared.

The patient made an uninterrupted recovery. Highest temperature after operation, 99.0° F.; highest pulse rate, 104; bowels well opened after calomel on third morning; stitches removed on tenth day; wound healed.

On October 12 a test meal was given; 150 c.c. were drawn off from stomach:—

Total acidity	59.0
Gmelin's reaction	absent	
Total chlorides	0.302
Free hydrochloric acid	0.007
Protein	0.164
Mineral	0.198

Patient left the hospital in excellent health on October 12; weight 9 st. 13 lb.

I did not see the patient again until August 26, 1908. She then told me that she had remained quite well until a month previously, when she began to suffer again from pain in the epigastrium, bearing no relation to food. Two days later the condition of the stomach was investigated. On passing a tube in the early morning the stomach was found to be empty. After a test meal 40 c.c. of gastric contents were obtained, containing a good deal of mucus and some blood. No lactic acid present.

Total chlorides	0.490
Free hydrochloric acid	0.032
Protein	0.317
Mineral	0.080

In view of the recurrence of hyperacidity and the presence of blood in the stomach I feared the recurrence of ulceration. When she left the hospital after operation I had advised her never to take meat again, a warning which I found she had disregarded. I again cautioned her as to her diet, and prescribed bismuth and sodium carbonate. The patient so strongly objected to the use of the stomach tube, and was so alarmed at the sight of blood in the gastric contents, that she discontinued attendance for some months, so I lost sight of her again, although I heard indirectly that she was better. In February, 1909, I persuaded her to come and see me again. She was then much better, although occasionally she suffered from attacks of pain, usually coming on during the night, accompanied by an acid taste in the mouth and not bearing any relation to food. Her weight was 10 st. 12 lb. There was no tenderness of the abdomen, the abdominal scar was soundly healed, and the patient looked in excellent health. Her pain was probably due to hyperacidity, and I could find no evidence of recurrence of ulceration. On bismuth and hydrocyanic acid she quickly improved and lost her pain, and I believe that if only she would be more careful in her diet she would remain well.

...

In a few of the recorded cases the total acidity was estimated before operation, and in still fewer the amount of free hydrochloric acid as estimated by Töpfer's method is noted. The estimation of free hydrochloric acid by this method, however, is very inaccurate, and the amount of total acidity includes both inorganic and organic acids. The important point, it appears to me, is the amount of free hydrochloric acid present in the gastric contents, and this is very well illustrated in the case I have related.

On admission into the hospital for the second time the free hydrochloric acid in the gastric contents was 0.072 per cent., more than three times the normal amount. The amount of free hydrochloric acid in the fluid escaping from the fistula—i.e., in the jejunal contents—was 0.044, or more than double the amount normally present in the stomach. This observation appears to me of considerable significance with regard to the aetiology of jejunal ulcers. Here we had evidence of the presence of a juice rich in free hydrochloric acid in a portion of the intestine in which the fluid is normally alkaline. That ulceration took place under these conditions is not surprising.

The analysis of the gastric contents after a week on a purely milk diet shows a very different state of affairs. The amount of free hydrochloric acid was then only 0.018 per cent., or just about normal, and the percentage of free hydrochloric acid in the fluid escaping from the fistula had diminished from 0.044 to 0.004. From these analyses we may, I think, draw the following inferences:—

(1) That the presence of an ulcer in the jejunum was connected with the large percentage of free hydrochloric acid present in the jejunal contents.

(2) That as the high percentage of free hydrochloric acid in the gastric contents rapidly diminished to a normal amount on a milk diet, the excess of free hydrochloric acid in this case was probably due to errors in diet.

(3) That the bile and pancreatic juices in the jejunum usually neutralize the free hydrochloric acid which normally escapes from the stomach. When, however, free hydrochloric acid reaches the jejunum in great excess, the alkaline juices are insufficient to neutralize all the free hydrochloric acid present.

Incidentally, I may point out that although after a week on a milk diet the amount of free hydrochloric acid had diminished by nearly two-thirds, the total acidity had diminished only from 85 per cent. to 80 per cent. This illustrates how little information Töpfer's method gives us as to the amount of inorganic acids present in the gastric contents.

ANTERIOR GASTROJEJUNOSTOMY FOR PYLORIC STENOSIS SUPPOSED TO BE MALIGNANT. RECURRENCE OF PAIN FIVE YEARS LATER. SEVEN YEARS LATER SEPARATION OF ADHESIONS ROUND JEJUNUM (? JEJUNAL ULCER). GOOD HEALTH UNTIL MARCH 1908; DEATH IN AUGUST 1908. MURPHY BUTTON STILL IN STOMACH.

Anterior gastrojejunostomy by means of a Murphy's button was performed by the late Mr. Walsham on a man, aged 69, suffering from pyloric stenosis, which was thought to be malignant. He remained well for nearly five years, but then began to suffer constant pain after food. He was sent to see me by Dr. Howard Distin, who thought that the pain was probably due to the irritation of the Murphy button, which, so far as was known, had never been passed. His weight was 8 st. 6 lb., a gain of 3 st. since the operation. The stomach was somewhat dilated, and just above the level of the umbilicus there was a rounded induration in the left rectus muscle. Under medical treatment the patient improved and remained well until the beginning of 1906, when he began to suffer severe pain in the left side, with occasional attacks of vomiting. I saw him again in July. He looked thinner and more careworn than when I last saw him, and he had lost 2 st. 4 lb. in weight. In the left rectus muscle there was a tender, nodular swelling about the size of a small tangerine orange. A test meal showed the presence of free hydrochloric acid, a trace of lactic acid, and a total acidity of 35. An X-ray photograph showed no trace of the button. After consultation with Dr. Fenwick it was decided to reopen the abdomen, in the belief that his symptoms were due to partial closure of the anastomotic opening, or to adhesions round the anastomosed loop.

On July 20 I reopened the abdomen. There were adhesions at the pyloric end of the stomach. The pylorus was markedly stenosed, but all trace of the former tumour had disappeared. The anastomosed loop of jejunum was firmly fixed to the anterior abdominal wall for a distance of 2 in. There was considerable induration in the jejunal wall, as if there existed, or had existed, an ulcer of the jejunum which had resulted in the adhesion of the jejunum to the abdominal wall. The separation of the adhesions to the abdominal wall would have involved an extensive operation, which I do not think justifiable in view of the patient's age. The other adhesions were divided and the wound sewn up.

The patient remained quite well until March, 1908, when his

appetite began to fail. He suffered no pain, but vomited occasionally. I saw him again in July. The stomach was then considerably dilated, marked stomach splash, no lump to be felt anywhere. It was apparent that the stomach was not emptying itself properly. With occasional lavage he improved for a time, but later his strength gradually failed, and he died in September, 1908, ten years after the gastrojejunostomy. Through the courtesy of Dr. Distin I was able to examine the abdominal viscera. On opening the abdominal cavity an interesting state of things was found. The stomach was enormously dilated and flabby. High up towards the cardiac end the button could be plainly felt loose in the cavity of the stomach. The anterior stomach wall had been drawn downwards and backwards, so that the site of the anastomosis was posterior rather than anterior. Although an anterior operation with a long loop had been performed the length of the afferent limb from the ligament of Treitz to the junction with the stomach now measured 4 in. only, and emerged from under the colon near the splenic flexure. Until a careful examination had been made it was difficult to believe that an anterior, and not a posterior, operation had been performed. This case bears out a view which I expressed some years ago, that after the anterior operation the afferent limb, although at the time of operation it is drawn up round the colon, becomes later displaced outwards so that it emerges from under the colon at or near the splenic flexure. Nature thus appears to protect the afferent loop from the influence of the excursions of the movable part of the transverse colon. The stomach was distended with formalin and examined at a later date.

When the stomach was opened¹ I found that the communication between the stomach and jejunum was much contracted and admitted only the tip of the little finger, and was surrounded with a considerable amount of indurated tissue. In the jejunum, just below the anastomotic opening, the mucous membrane was puckered, as if the site of an old ulcer. Microscopical examination showed old chronic inflammation. The pylorus was much stenosed and indurated, and its lumen was diminished to the size of a cedar pencil.

I have seen in consultation two other probable cases of jejunal ulcer, both in patients on whom the posterior operation had been

¹ This examination was made too late for the case to be included in my statistics. I have little doubt that at one time there was a jejunal ulcer, but as the existence of an open ulcer was not demonstrated it is perhaps as well that the case should be included among the "doubtful cases."

performed. Details of one of these cases are given among the doubtful cases.¹

THE FREQUENCY OF JEJUNAL ULCER.

Jejunal ulcer following gastrojejunostomy is distinctly uncommon. The first case was reported by Braun [1] in 1899. Four years later Brodnitz [3] collected fifteen cases, and in 1905 Tiegel [21] collected twenty-two. Gosset [6] in 1906 published an admirable article with an abstract of thirty-one cases. In 1907 Schostak [17], in an exhaustive dissertation on this subject, summarized thirty-one cases, and added details of four cases previously unreported. Last year Einar Key [4] published a full account of six additional cases, one of which is of especial interest, as it is the first reported instance of jejunal ulcer following gastrojejunostomy for gastric carcinoma. In my Hunterian Lectures [13], published in 1906, I related two unpublished instances of jejunal ulcer, and mentioned that jejunal ulcer had occurred in three of 295 patients whose after-history I had been able to ascertain. Since that time I have followed up the late history of an additional fifty-three patients, and in none of these has jejunal ulcer occurred. It is doubtful whether all the cases recorded as instances of jejunal ulcer were really such, as in some of them the diagnosis was a matter of supposition rather than of demonstration. Such cases—and to this class belongs one of the cases I now publish—are not included in my statistics, although I have given brief details of them under the heading of "Doubtful Cases." In this paper I give details of fifty-eight cases already published by other writers, so that, including my own two cases and the two I reported in 1906, fifty-two certain and eleven doubtful cases are now on record, a total of sixty-three cases.²

The proportion of cases in which gastrojejunostomy is followed by jejunal ulcer is impossible to estimate with accuracy. Mikulicz had two cases in 160 gastrojejunostomies. Schostak reports that in the

¹ In one of these cases the diagnosis has since been verified by operation. This patient, whom I saw with Dr. Hort and Mr. Cheate, was subsequently operated on by Mr. Moynihan, who has courteously informed me that he found a duodenal ulcer and two jejunal ulcers.

² At the German Congress of Surgeons (*Verhandl. der Deutsch. Gesellsch. f. Chir.*, 1906, i., p. 78) Kelling stated that he had observed one instance of jejunal ulcer after gastrojejunostomy ("En-Y"), and Gosset in his paper mentions a case of Eiselsberg's; but, as no details of these cases are available, I have not included them in my paper. Several of the cases have been reported by more than one writer; and if cases of which no details are given are included in statistics, there is a risk that they may be counted twice over. If these two cases be included the number of cases recorded is now sixty-five.

Krönlein Clinic one instance of jejunal ulcer occurred in ninety-two gastrojejunostomies for non-malignant disease of the stomach. Wickenhauser observed three jejunal ulcers in 115 gastrojejunostomies, and Rotgans one in forty-nine. As I have already mentioned, jejunal ulcer occurred in three of 348 patients whose after-history I traced. Some cases doubtless have not been recorded, and probably more have been unrecognized; but supposing that these equal in number those published, even so the total number would be small in comparison with the enormous number of times the operation of gastrojejunostomy has been performed. Probably more gastrojejunostomies have been performed in America than in any other country, but so far only three cases of jejunal ulcer have been published in American surgical literature. When I visited the United States two years ago I made inquiries of the many surgeons I had the pleasure of meeting as to their experience of this complication. Dr. W. J. Mayo and Dr. C. H. Mayo, who perform some sixty to eighty gastrojejunostomies annually, have yet to observe a case of jejunal ulcer after gastrojejunostomy. Dr. J. B. Murphy has not met with a case, and other leading surgeons testified to a like experience. In the present state of our knowledge we are, I think, justified in estimating the probable risk of jejunal ulcer following gastrojejunostomy at under 2 per cent.

Clinically the cases of jejunal ulcer hitherto recorded are divisible into two groups:—

Group I.—Cases in which perforation into the general peritoneal cavity occurs. In this group are nineteen cases.

Group II.—Cases in which, owing to the formation of localizing adhesions, perforation does not result in the escape of bowel contents into the general peritoneal cavity. This group comprises thirty-three cases and includes two subdivisions: (a) Cases in which the base of the ulcer becomes adherent to the abdominal parietes, so that perforation results in inflammatory exudation into the abdominal wall (twenty-eight cases). (b) Cases in which the base of the ulcer becomes adherent to and perforates into a hollow viscus—the colon in the cases so far recorded (five cases).

I think it would be well to drop the use of the word "peptic" in connexion with jejunal ulcers. It is quite unnecessary and is misleading, as it implies an hypothesis which, as I shall try to show later, is incorrect.

As I shall have occasion to point out later, in a considerable proportion of the cases recorded as jejunal ulcer, the ulcer was situated at the site of

the anastomosis, and therefore was gastric as much as jejunal. I think that ulcers in this situation should be clearly distinguished from jejunal ulcers proper, and I propose so to distinguish them by describing them as gastrojejunal ulcers.

At this point I will give a brief abstract of all the cases so far recorded which I have been able to find.

RECORDED CASES OF JEJUNAL AND GASTROJEJUNAL ULCER AFTER
GASTROJEJUNOSTOMY.

*Group I.—Cases in which Perforation ensued into the General Peritoneal
Cavity (Nineteen Cases).*

(1) BRAUN, *Kongressbericht*, 1899, ii, p. 94.—Male, aged 25. Posterior gastrojejunostomy by suture in November, 1897, for pyloric stenosis. Some days after operation vomiting recurred, but gradually ceased again. Symptoms recurred soon after patient left hospital, and early in 1898 patient was readmitted. Stomach was found to be dilated below the umbilicus; patient improved under treatment. In October, 1898, eleven months after the operation, patient had violent pain in the stomach, accompanied by vomiting. Two days later he was admitted into hospital with signs of peritonitis and died the following night. Post mortem: Peritonitis: perforated jejunal ulcer in efferent limb, 1 in. below anastomosis; anastomotic opening large.

(2) HAHN, *Kongressbericht*, 1899, p. 74.—Male. Anterior gastrojejunostomy for pyloric stenosis. One year later, while lifting a heavy weight, patient was seized with severe pain in the abdomen; death within twenty-four hours. Post mortem: Perforated ulcer in efferent jejunal limb, 1 to 2 in. below anastomosis.

(3) KÖRTE, *Kongressbericht*, 1900, p. 137.—Male, aged 30. Anterior gastrojejunostomy in February, 1897, for pyloric stenosis. Remained well until March 15, 1900, when he had sudden pain in the abdomen with constipation. On March 20 laparotomy was performed. General peritonitis below the transverse colon, considered to be of appendicular origin. The appendix was removed, and the patient died on the following evening. Post mortem: A subphrenic abscess was found which had ruptured into the general peritoneal cavity. The cause of this abscess was a jejunal ulcer, 7 cm. from the stoma, which had perforated the intestinal wall as far as the serous covering. On the serous surface over the ulcer was a purulent exudation. The original duodenal ulcer was completely healed.

(4) STEINTHAL, *Kongressbericht*, 1900, p. 139.—Male, aged 44. Posterior gastrojejunostomy, by means of Murphy's button, for pyloric stenosis. Death ten days later. Post mortem: Peritonitis due to perforation of jejunal ulcers, two in the afferent and two in the efferent limb of the jejunum. It was

thought that the predisposing cause of the perforation was the atheromatous condition of the blood-vessels.

(5) GOEPEL, *Kongressbericht*, 1902, p. 108.—Anterior gastrojejunostomy was performed on a man suffering from pyloric stenosis. Thirteen months later the patient died from peritonitis. Post mortem: A perforated ulcer of the jejunum, 2 to 3 mm. from the anastomotic opening.

(6) GOEPEL (*loc. cit.*).—In this case the patient, a man, was suddenly seized with abdominal pain and vomiting four months after anterior gastrojejunostomy for pyloric stenosis. The abdomen was opened five hours later, and a perforated jejunal ulcer 2 to 3 mm. from the anastomotic opening was found and sutured. The patient recovered.

(7) GOEPEL, quoted by Gosset, *Revue de Chirurgie*, 1906, p. 300.—Male, aged 34. Anterior gastrojejunostomy was performed, and after nine months of good health a jejunal ulcer perforated. The patient recovered after prompt laparotomy and suture of the ulcer.

(8) MIKULICZ, quoted by Tiegel, *Mitteilungen aus den Grenzgebieten der Medizin und Chirurgie*, vol. xiii, p. 897.—A female infant, aged 2 months. Anterior gastrojejunostomy with entero-anastomosis for infantile pyloric stenosis. The infant progressed well for two months, but then passed two black motions. The following morning she vomited a large quantity of blood, and died two weeks later. Post mortem: A perforated ulcer in the ascending limb of the jejunum, and several other ulcers in the jejunum in the neighbourhood of the anastomosis.

(9) GEO. H. EDINGTON, *Glasgow Medical Journal*, June, 1907.—In September, 1899, anterior gastrojejunostomy was performed on a man, aged 39, for dilated stomach with epigastric pain. He remained well until July 1904, when he began to suffer from vomiting, with severe pain in the right hypochondrium. Cholecystotomy was performed and a stone extracted from the cystic duct. He remained well until November 9, 1906, when he was seized with epigastric pain. On the following day a diagnosis of perforated gastric ulcer was made and the abdomen opened. The abdomen was found to be full of muddy fluid, and a perforation existed on the anterior surface of the jejunum immediately to the left of the anastomotic opening and on the descending limb. The perforation was closed, but the patient never rallied and died three and a half hours later.

(10) BASIL HALL, communicated to me by Dr. Basil Hall. A brief abstract was published in "Gastric Surgery," 1906, p. 137.—Gastrojejunostomy by the supra-colic route was performed in May, 1901, on a man aged 48. This patient was seen at the end of the year 1902, and was then in excellent health and had put on over 3 st. in weight. In June, 1903, whilst drunk, he was seized with signs of perforation, and died next day. He was known to be a heavy drinker. Post mortem: A perforated jejunal ulcer was found 6 in. below the anastomosis.

(11) RUSHTON PARKER. Notes of this case were kindly sent to me by Mr. Rushton Parker. I gave brief details of the case in "Gastric Surgery" (1906), p. 137.—Pyloroplasty was performed in December, 1900, on a man, aged 51, suffering from pain and vomiting. After a short period of relief the symptoms returned, and a year later anterior gastrojejunostomy was performed. The patient was seen in February, 1904, and was then in excellent health. At 7 a.m. on August 13, 1904, he was moving some object, not a heavy weight, when he had sudden pain and became collapsed. He was seen at 5 p.m. by Mr. Dean, of Lancaster, but was then too ill for any operation, and he died a few hours later. Post mortem: Abdominal cavity full of fluid; intestines adherent with lymph. In the jejunum opposite to the anastomotic opening was a perforation the size of a shilling-piece. The ulcer was an old one with thickened edges. The pylorus would hardly admit the tip of a pair of scissors, and was as hard as cartilage.

(12) HAMANN, *Cleveland Medical Journal*, May, 1907, p. 183.—Anterior gastrojejunostomy with entero-anastomosis was performed on a man, aged 48, who had symptoms of gastric ulcer. A Murphy's button was employed for both anastomoses. The smaller button was passed on the eleventh day. The patient progressed well until the twenty-second day after the operation, when he complained of pain in the epigastrium, while an indistinct tender mass was felt in the epigastric and left hypochondriac regions. Death occurred suddenly twenty-six days after the operation. Post mortem: General peritonitis. The gastro-intestinal union was perfect. On the anterior wall of the efferent limb of the jejunum, just beyond the anastomosis with the stomach, there was a perforated ulcer about 1 in. in diameter, with a sharply marked border. The button was *in situ*, but it was not considered that this was the cause of the ulcer.

(13) BATTLE, *Lancet*, vol. ii, p. 1246, and p. 274, 1906, and private communication.—Anterior gastrojejunostomy, by means of a Murphy's button, was performed in July, 1904, on a man, aged 30, suffering from vomiting due to pyloric obstruction. Some months later the abdomen was reopened on account of intestinal obstruction due to the button, which was removed. Twenty-two months after the gastrojejunostomy the patient was readmitted into the hospital with signs of perforation. Four hours after the onset of the symptoms the abdomen was opened, and a small perforated ulcer $\frac{1}{2}$ in. in diameter was found immediately below the anastomotic opening. The surrounding peritoneum was reddened and covered with flakes of lymph. Invagination of the ulcer with flushing and drainage was followed by recovery. Mr. Battle has been good enough to inform me that the patient reported himself a month ago (March, 1908) as being in excellent health.

(14) BATTLE (loc. cit.).—A woman, aged 37, was admitted into St. Thomas's Hospital in March, 1903, with signs of perforation. The abdomen was opened, and a perforated ulcer was found on the anterior wall of the stomach near the pylorus. This was sutured and the patient recovered. Later she showed signs of pyloric obstruction, and accordingly an anterior gastrojejunostomy was

performed in April, 1904. On May 5, 1905, she was seized with severe pain, and on the following day the abdomen was reopened and a perforated jejunal ulcer was found $1\frac{1}{2}$ in. from the anastomosis. This was sutured and the patient recovered. On March 14, 1905 (?1906), the patient was again admitted with signs of perforation, and on opening the abdomen a perforation was discovered at the junction of the stomach and the jejunum. The perforation was closed by suture and the patient recovered, and is now (March, 1908) in good health.

(15) GRASER, *Verhandlung der deutsche Gesellschaft für Chirurgie*, 1906, i, p. 95.—Posterior gastrojejunostomy, by means of a Murphy's button, was performed in 1902 by Dr. Dörfler, at Wissenberg, on a woman suffering from a pyloric tumour which was thought to be inoperable carcinoma. About Christmas time in 1905 vomiting recurred. The patient died from hæmorrhage and peritonitis in the following May. Post mortem: An ulcer of the jejunum at the site of anastomosis was found. In the base of the ulcer was an eroded artery. The anastomotic opening was narrowed so that it only admitted a cedar pencil.

(16) HYBRINETTE, reported by Einar Key (loc. cit.).—In January, 1899, anterior gastrojejunostomy "En—Y" was performed on a woman, aged 45, who had a crater-like ulcer adherent to the pancreas on the posterior wall of the stomach. March, 1906: Patient readmitted. Occasionally she has had severe abdominal pain. During this month the pain has been more constantly present and more severe than before. After lavage on March 16 sudden severe abdominal pain; abdomen distended, diffuse tenderness. The abdomen was reopened; gas escaped on opening the peritoneal cavity. Flakes of fibrin in neighbourhood of old gastrojejunostomy. On the right a considerable amount of turbid exudation free in abdominal cavity. On separating adhesions a perforated ulcer, partly covered by omentum, was discovered at the side of the gastrojejunostomy. The ulcer had constricted the site of the gastrojejunostomy; the edges of the ulcer were excised and the communication between the stomach and intestine was enlarged; the peritoneal cavity was thoroughly flushed. April 21: The patient was discharged; total acidity, 50. June, 1906: No more vomiting, but suffers from pain in the pit of the stomach and acid eructations.

(17) LENNANDER, reported by Einar Key (loc. cit.).—On December 2, 1906, partial gastrectomy and anterior gastrojejunostomy were performed on a woman, aged 25, suffering from cancer of the stomach. Ten days later the patient died from general peritonitis due to perforating ulcers in the coil of intestine leading to the site of anastomosis.

(18) CACKOVIC, *Liecnicki viestnik*, 1903, No. 7.—In this case posterior gastrojejunostomy was performed on a man, aged 30. Five days after the operation the patient died. At the post mortem four perforated ulcers were found in the efferent limb of the jejunum. One of the ulcers was opposite the anastomotic opening, and one 15 cm. below it.

(19) DELALOYE, *Deutsche Zeitschrift für Chirurgie*, 1906, p. 518.—Anterior gastrojejunostomy was performed in October, 1896, on a man, aged 41, who had suffered from dyspepsia since the age of 15. In the beginning of 1900 the patient was seized with sudden pain in the region of the stomach. Under dietetic treatment he improved, but in June, 1902, he had a second attack and died two days later. A fist-size swelling was felt to the left of the middle line. At the post mortem a perforated ulcer was found at the site of the anastomosis, and round afferent jejunal limb a mass of adhesions adherent to the anterior abdominal wall. It was considered probable that an ulcer existed at the time of the first attack, but that perforation was at that time prevented by the formation of protective adhesions.

Group II.—Cases in which owing to the Formation of Adhesions General Peritonitis did not ensue.

(A) CASES IN WHICH ULCER WAS ADHERENT TO ABDOMINAL PARIETES (TWENTY-EIGHT CASES).

(1) MIKULICZ, *Kongressbericht*, 1899, p. 74.—Male, aged 32. Anterior gastrojejunostomy in September, 1898, for pyloric stenosis. Patient improved for a time, but four months after operation he returned to the hospital with a return of his symptoms. Total acidity of gastric contents, 90 to 110. In January, 1899, the abdomen was reopened and a perforated ulcer was found at the anastomotic junction, the ulcer being partly in the stomach and partly in the jejunum. It was adherent to the anterior abdominal wall. The perforation was sutured and an entero-anastomosis performed. After a short interval the pain recurred. In July the abdomen was reopened, and again an ulcer was found in the jejunum at the level of the gastrojejunostomy. Part of the ascending limb of the jejunum was resected; Doyen's operation performed. January, 1900: Abdomen reopened, and after separating adhesions two nodes were found in the neighbourhood of the pylorus, probably indicating ulcers. Jejunostomy by Witzel's method was performed. In February the patient returned to hospital suffering from severe pain in the sacral and hepatic regions, accompanied by the passing of black stools. Gastric acidity 60 to 72. In March the abdomen was reopened and the vessels and nerves going to the greater and lesser curvatures were ligatured. In spite of this procedure the pain persisted. Later the patient's condition improved somewhat, but he had to resort to frequent stomach lavage and the use of morphia.

(2) MIKULICZ, recorded by Tiegel, *Mitteilungen aus den Grenzgebieten der Medizin und Chirurgie*, xiii, p. 897.—Male, aged 25. July, 1899: Anterior gastrojejunostomy with entero-anastomosis for pyloric stenosis. October 1: Sudden giddiness, followed by vomiting of several pints of dark material. With a month's rest in bed on a milk diet the patient's condition improved. January, 1901: Pain again became severe, especially at nights. After a test meal,

230 c.c. of fluid were drawn off—total acidity 64, free hydrochloric acid 53. A few days later the abdomen was reopened. The stomach was found to be fixed to the anterior abdominal wall by adhesions in the region of the gastrojejunostomy. On separating the adhesions a perforated jejunal ulcer was discovered in the ascending jejunal limb. The anastomosis between the stomach and jejunum admitted three fingers, and the entero-anastomotic opening two fingers. After excision of the ulcer, jejunostomy by Witzel's method was performed. Following this operation the patient's condition immediately improved and he put on weight. After three months the fistula was allowed to close. In October, 1901, the patient was readmitted into the hospital with well-defined and localized pain. Three-quarters of an hour after a test meal 18 c.c. of fluid were drawn off; total acidity 45, free hydrochloric acid 32. A third laparotomy was performed. After separation of adhesions an ulcer was found just above the site of the entero-anastomosis. During the next few days the patient vomited much dark material. A week after operation the abdomen was reopened in the belief that the excision and suture of the ulcer had resulted in obstruction. A new entero-anastomosis was made and gastrojejunostomy by means of a Murphy's button. This latter proved a very difficult procedure. Ten days later the patient died from peritonitis, the result of perforation of the stomach at the site of the button.

(3) QUÉNU, *Bulletins et Mémoires de la Société de Chirurgie de Paris*, 1902, xxviii, p. 250, and also 1904, xxx, p. 194, and private communication.—Male, aged 29, July, 1897: Anterior gastrojejunostomy by Murphy's button for pyloric stenosis. Towards the close of the year 1898 the patient began to have sour eructations, and two or three times in the month attacks of vomiting. Occasionally he passed black motions. In June, 1901, for the first time since his operation, the patient again began to suffer pain in the umbilical region, and having no relation to the ingestion of food. In October, 1901, the patient looked in good health, and did not appear to have lost flesh. In the sheath of the right rectus muscle, close to the umbilicus, an induration measuring 2 in. by 3 in. was felt. It was tender to palpation. In January, 1902, the abdomen was reopened. The anastomosis was adherent to the abdominal wall. On separating adhesions, a jejunal ulcer was discovered communicating with a cavity in the substance of the right rectus muscle. The gastro-anastomotic opening admitted the thumb and index finger. The anastomosed loop was resected, together with the muscle adherent to it. An anterior gastrojejunostomy "En—Y" was then performed. The patient remained well for six months, and then had recurrence of his symptoms. Gastric analysis showed hyperacidity. In June, 1903, the abdomen was reopened. Many adhesions were present. A jejunostomy "En—Y" was performed.

Dr. Quénu has kindly informed me (February, 1909) that the patient's pain has disappeared since the last operation, and no further ulceration has occurred.

(4) KOCHER, *Kongressbericht*, 1902, p. 103.—The patient was a man on whom gastrojejunostomy "En—Y" was performed for pyloric stenosis. After

remaining well for three months he again began to suffer pain, which was especially severe about two hours after taking food. Later, a swelling of the abdominal wall in the region of the left rectus muscle was discovered, and examination of the gastric contents showed a diminution of hydrochloric acid. Professor Kocher thought that at the time of the original operation he had overlooked a carcinomatous growth. One year after the gastrojejunostomy the abdomen was reopened, and a perforated jejunal ulcer was found. This, together with the surrounding indurated tissue, was resected, and the patient made an uninterrupted recovery.

Dr. Albert Kocher has kindly informed me that this patient died some years later from arteriosclerosis; but to the time of his death he had suffered from no further gastric trouble, and examination showed that his stomach was performing its functions well.

(5) HEIDENHAIN, *Kongressbericht*, 1902, p. 108, and personal communication.—The patient was a man, aged 48, on whom in the year 1898 anterior gastrojejunostomy was performed for pyloric stenosis and a bleeding ulcer. In May, 1899, the patient returned, suffering severe pain. In the left rectus muscle there was a marked induration. On opening the abdomen a perforated jejunal ulcer was found. The perforation was closed and the patient recovered, but some months later his symptoms returned. The abdomen was reopened and a perforated jejunal ulcer adherent to the left rectus muscle was found at the upper margin of the anastomotic opening. The perforation was closed. After this operation the stomach did not appear to be emptying itself well, so a new gastrojejunostomy opening was made. Four years later the patient was reported to be in good health.

Professor Heidenhain has very courteously given to me the following after-history. For the past seven years the patient has not required to consult a physician. He is perfectly well except for chronic constipation, which he relieves by enemata. He has no gastric pain, no sickness, and very seldom eructations. He takes almost any kind of food. Examination shows that the stomach is large. The patient states that his stomach is always empty in the morning.

(6) KRÖNLEIN, *Kongressbericht*, 1902, p. 110; fuller report by Schostak, *Beiträge für klinische Chirurgie*, 1907, lvi, p. 361.—Anterior gastrojejunostomy was performed in October, 1895, on a man, aged 36, who had suffered from gastric pain with hæmatemesis for eight months. At the operation was found marked induration of the head of the pancreas, probably the result of duodenal ulcer, which had led to pyloric obstruction. In the spring of 1897 the patient returned with a hard, tender swelling of the abdominal wall, and complaining of severe pain in the left side one to two hours after food. After a time both swelling and pain disappeared, only to return again in the autumn of the same year. In July, 1901, there was below the navel on the left side a hard, tender swelling the size of a fist, adherent to the abdominal wall. A diagnosis of peptic jejunal ulcer was made. The abdomen was reopened and a perforated jejunal ulcer, which was adherent to the abdominal

wall, was sutured. For a time the patient remained well, but again began to suffer from gastric pain and vomiting. Towards the end of 1902 the gastric pain was especially severe, and he brought up a quantity of blood. He continued at work on and off until July, 1905. In August, 1905, by which time he was reduced to an extreme degree of exhaustion and inanition, the abdomen was reopened. The stomach was greatly dilated and much adherent, especially in the neighbourhood of the gastrojejunostomy. The anastomotic opening was completely obliterated. No sign of a peptic ulcer could be discovered. Posterior gastrojejunostomy was performed, an entero-anastomosis being made between the afferent limb of the new and the efferent limb of the old gastrojejunostomy. In December, 1906, the patient remained quite well, and there was no suggestion of a recurrence of his gastric troubles.

(7) CZERNY, *Beiträge für klinische Chirurgie*, xxxix, p. 98 (supplement).—In the year 1893 Czerny performed posterior gastrojejunostomy on a man, aged 59, who had pyloric stenosis. He remained well for eight years. Then he began to suffer pain in the gastric region. Marked dilatation of the stomach was present and hyperacidity, and a resistance in the neighbourhood of the umbilicus. Nine years after the first operation the abdomen was reopened, and an ulcer was found at the level of the anastomosis. After separating adhesions, the ulcer, together with part of the jejunum, was resected, and a gastrojejunostomy "En—Y" (posterior) was performed. The ascending limb was joined to the descending limb by means of a Murphy's button. The patient died four days later of peritonitis.

(8) CZERNY, *Beiträge für klinische Chirurgie*, xxxix, p. 99 (supplement).—This patient was a man, aged 42, on whom posterior gastrojejunostomy by means of a Murphy's button was performed for pyloric stenosis in 1900. Six months later his symptoms recurred and a gastro-enteroplasty at the site of the anastomosis was performed. Nine months later he began to suffer from severe attacks of pain. The abdomen was opened anew and a peptic ulcer was found at the level of the anastomosis adherent to the posterior abdominal wall. The pylorus was much stenosed and was the site of an ulcer. The pylorus, with part of the duodenum and of the stomach, was excised, and the cut ends united by suture. Six days later the patient died from peritonitis.

(9) NEUMANN, *Deutsche Zeitschrift für Chirurgie*, lviii, p. 270.—In March, 1897, antero-gastrojejunostomy was performed on a man, aged 24, who had suffered from stomach trouble for six years. In May, 1897, an antero-anastomosis was performed on account of regurgitant vomiting. Some months later the patient began to suffer from colicky pain after food, and a hard tender swelling formed to the right of the umbilicus. In May, 1898, the swelling was incised, and a fistula leading into the stomach was found; the fistula was scraped and gradually healed up. The patient remained well for a time, but then the symptoms recurred. In November, 1899, the abdomen was reopened. The jejunum was separated from the stomach. The anastomosed loop was adherent to the abdominal wall, and a perforated jejunal ulcer was discovered.

This was resected. He remained well for six months, but in July, 1900, another fistula formed. An ulcer in the region of the anastomosis was found. As the stomach was not dilated, and as the pylorus admitted a finger, the gastrojejunostomy opening was closed. The patient left the hospital in perfect health.

(10) MIKULICZ, *Boston Medical and Surgical Journal*, No. 23, 1903.—In October, 1893, anterior gastrojejunostomy with entero-anastomosis was performed on a man, aged 53, for pyloric stenosis. The anastomosis was made 28 in. below the duodenum, and the entero-anastomosis 4 in. lower down. The patient returned in January, 1903, stating that he had had four attacks of hæmatemesis—the first in June, 1899, and the last in November, 1902. At this time a tender induration in the right rectus muscle was noticed. Hyperacidity and hypersecretion were present. In February, 1903, the abdomen was reopened, and, after cutting through much indurated vascular tissue, a perforating ulcer half in the stomach and half in the jejunum was found. The perforation was sutured transversely by two rows of suture. Six days later the patient died of peritonitis.

(11) MIKULICZ, quoted by Tiegel (loc. cit.).—In January, 1899, anterior gastrojejunostomy with entero-anastomosis was performed on a man, aged 33, who had suffered from gastric troubles for many years. He remained well for a year and a half, and then had recurrence of his symptoms. In 1901 a second operation was advised. In reopening the abdomen the stomach, which was adherent to the abdominal wall, was opened, so the operation was not proceeded with further. In March, 1903, he came under the care of Mikulicz. To the left of the umbilicus was a tender swelling. Three quarters of an hour after a test breakfast, consisting of 400 c.c. of oatmeal soup, 115 c.c. of fluid were drawn off; total acidity 55, free hydrochloric acid 47. Five hours after a test meal (hashed beef 125 grm. some bread and water) 130 c.c. of fluid were drawn off; total acidity 62, free hydrochloric acid 45. On March 16, 1903, the abdomen was reopened. Extensive adhesions were found, involving both the stomach and intestine. These adhesions surrounded a perforated jejunal ulcer, which was sutured transversely. For six months the patient remained perfectly well. He then began to suffer pain in the neighbourhood of the scar, radiating to his back. A tender, hard swelling formed in the region of the cicatrix. The pain bore no relation to food, and after medical treatment gradually diminished and disappeared; during this period, which lasted about six weeks, the patient lost 10 lb. in weight. Four hours and a half after a test meal 230 c.c. of fluid were drawn off; total acidity 42, free hydrochloric acid 22. In December, 1903, 128 c.c. of fluid were drawn off from the fasting stomach; total acidity 22, free hydrochloric acid 9. After a test breakfast 241 c.c. were drawn off; total acidity 20, free hydrochloric acid 10. After a test meal, 240 c.c.; total acidity 75, free hydrochloric acid 38.

(12) SCHLOFFER, *Beiträge zur klinische Chirurgie*, 1902, xxxii, p. 357, and private communication.—In July, 1900, anterior gastrojejunostomy was performed on a woman, aged 31, suffering from pyloric stenosis. Three days

later an entero-anastomosis was performed on account of regurgitant vomiting. In October, 1900, the patient was again in hospital, complaining of severe pain in the region of the stomach. To the left of the operation scar was felt a small hard swelling. The abdomen was reopened and a swelling found adherent to the stomach and to the anterior abdominal wall, involving the omentum and hiding the entero-anastomosis from view. As both gastro-jejunoscopy and entero-anastomotic openings were patent, nothing further was done. In August, 1901, the patient was in excellent health.

Professor Schloffer has kindly furnished me with the following details as to the after-history of this patient: The patient was admitted into hospital for the third time on March 31, 1902. She had again suffered from severe pain in the region of the stomach, and had noticed a very tender swelling in the region of the navel. Examination showed a hard, tender swelling, the size of a fist, in the middle line above the navel, with reddening of the overlying skin. On April 18 the abdomen was reopened. The site of the former gastrojejunoscopy was occupied by a large tumour, and the gastrojejunoscopy opening was probably obliterated. Accordingly a new anterior gastrojejunoscopy was performed in the portion of stomach between the pylorus and the former gastrojejunoscopy. When the patient left the hospital, on May 13, the swelling so plainly felt before operation had completely disappeared. The patient was admitted into hospital for the fourth time on November 27, 1902. Since the last operation the patient had suffered occasionally from pain, and sometimes from vomiting, but latterly less frequently. Six weeks before admission she was seized again with severe pain, and accompanied every two or three days by vomiting. No hæmatemesis or melæna. Examination showed a round, hard, tender swelling about the size of an apple situated on the right side beneath the costal arch. On December 11, 1905, the abdomen was reopened. Stomach not dilated. Pyloric portion of stomach fixed to the tumour mentioned above. A posterior gastrojejunoscopy was performed. To this gastrojejunoscopy the afferent loop of the first gastrojejunoscopy was anastomosed. In March, 1908, the patient was in excellent health.

(13) BRODNITZ, *Kongressbericht*, 1903, p. 77, and private communication.—Anterior gastrojejunoscopy for pyloric stenosis was performed in 1899 on a man aged 58. After three years and nine months of perfect health he began to suffer from severe attacks of pain in the region of the stomach. In the cicatrix was a hard, tender swelling, which in the course of a month increased considerably in size. On opening the abdomen and separating adhesions a jejunal ulcer was discovered. A second ulcer was found at the posterior border of the anastomosis. A portion of the stomach wall, together with part of the ascending and descending jejunal loops, was resected, and a gastro-jejunoscopy "En—Y" was performed. On the nineteenth day after operation a fistula formed, from which liquids ingested escaped. The fistula gradually closed up. He remained well for five months, when the pain recurred and a tender infiltration of the left rectus muscle was found. Free hydrochloric acid 1·8 per 1,000. Under medical treatment the pain diminished, and the swelling was no longer tender to palpation.

Dr. Brodnitz has kindly furnished me with the following later details respecting this patient: After several months in perfect health, early in the year 1904 the patient began to have pain in the stomach region. The infiltration of the left rectus at the navel, which had never disappeared, became markedly tender, the tissues were reddened, and it seemed as if a hernia were forming. A Witzel's jejunostomy was performed. The patient was fed entirely through the fistula, but the pain continued, and the patient vomited quantities of clear acid fluid. After three weeks the patient was again fed by the mouth, and copious doses of magnesia were administered. After three months the inflammatory effusion, which had gradually spread over the whole gastric region, slowly subsided. At the present time (January 18, 1908) the patient appears blooming, and is able to follow his employment. He can eat anything, but has to take regular doses of magnesia to prevent a feeling of oppression in the stomach. In the left rectus, above the level of the navel, is a thick, painless infiltration, which one not knowing the history of the case would regard as a malignant tumour invading the abdominal wall.

(14) MAYO ROBSON, *Medico-Chirurgical Transactions*, 1904, lxxxvii, p. 339. —In January, 1900, anterior gastrojejunostomy was performed on a man, aged 44, suffering from gastric pain and hæmorrhage. The patient remained well for two years, regained his normal weight, and was able to do his work. He then began to have pain in the epigastrium, having no relation to food. At times the pain was very acute. There was marked tenderness in the epigastrium, especially on the left of the middle line. On several occasions there was dark blood in the motions. In May, 1903, there was a well-marked tender swelling in the epigastrium to the right of the middle line. On reopening the abdomen the stomach was found adherent to the abdominal wall, and on separating adhesions a perforated ulcer was found involving the whole circumference of the jejunum at its junction with the stomach. The ulcerated portion of jejunum was resected and a gastrojejunostomy "En-Y" performed.

(15) KRAUSE, reported by Jahr, *Berliner klinische Wochenschrift*, 1905, No. 44a, p. 100, and private communication. —In December, 1903, anterior gastrojejunostomy with entero-anastomosis was performed on a man, aged 29, who had suffered from gastric troubles for five years. In August, 1904, he began to suffer from pain in the left hypochondrium radiating to the back. The pain increased in severity, necessitating his readmission into the hospital. At the seat of the pain was a tender, flat induration. From the fasting stomach 90 c.c. of fluid were drawn off; total acidity 45, free hydrochloric acid 25. After a test meal 200 c.c. of stomach contents were drawn off; total acidity 55, free hydrochloric acid 32. As medical treatment had been of no avail, the abdomen was reopened in September, 1905. A perforated jejunal ulcer was discovered opposite to the anastomosis and adherent to the anterior abdominal wall. The ulcer was excised and the opening closed with three rows of sutures.

Professor Krause has courteously informed me that this patient was seen and examined on February 8 of this year: "He is in good health. Much food remains in fasting stomach. Free hydrochloric acid present, no lactic acid. Test meal 100 c.c. well digested; total acidity 43, free hydrochloric acid 25."

(16) VON HACKER, reported by Hofmann, *Beiträge zur klinische Chirurgie*, 1906, I, p. 736.—Anterior gastrojejunostomy with entero-anastomosis was performed on a woman aged 22. The patient remained well for six years, and then began to suffer from a feeling of oppression to the left of the navel, in which region there was a clearly palpable resistance. Free hydrochloric acid 0.9 per mille, total acidity 1.8 per mille. Six days later the abdomen was reopened (June 5, 1905). Many adhesions were separated and a mass of scar-tissue excised, revealing a circular peptic jejunal ulcer. This was excised and the opening closed by suture. Ten months later the patient remained in good health.

(17) VON HACKER, reported by Hofmann (loc. cit., p. 742).—Posterior gastrojejunostomy was performed in June, 1899, on a man, aged 45, suffering from pyloric stenosis, which was considered to be malignant. The patient remained well for one year, and then pain and vomiting recurred and gradually increased so that in 1904 a further operation was decided on. Free hydrochloric acid 1.46 per mille, total acidity 1.84 per mille. In July, 1904, the abdomen was reopened. At the site of the former gastrojejunostomy was found an ulcer-tumour the size of an egg. A new posterior gastrojejunostomy nearer the pylorus was performed by means of a Murphy's button. One and a half years later the patient remained quite well.

(18) KRÖNLEIN, reported by Schostak, *Beiträge für klinische Chirurgie*, 1907, lvi, 2, p. 366.—Anterior gastrojejunostomy was performed in March, 1903, by Dr. Aeppli on a man, aged 22, suffering from pyloric stenosis. After the operation the patient remained quite well for a year and put on 40 lb. in weight. In the course of the year 1904 he again suffered from severe abdominal pain, and he noticed a hard, tender swelling, about the size of an egg, to the left of the navel. Under medical treatment he improved for a time. In May, 1905, he was admitted into hospital under Professor Krönlein, who made a diagnosis of peptic jejunal ulcer. The total acidity of the stomach contents was 56. In June, 1905, the abdomen was reopened. The stomach was greatly dilated and bound down by adhesions. On separating these an opening was found at the upper part of the anastomosis, involving both the stomach and jejunum. The base of the ulcer was formed by the abdominal wall in the region of the left rectus muscle. The perforation was closed by suture and an omental graft sewn over it. An entero-anastomosis was performed between the afferent and efferent loops of the jejunum. All symptoms disappeared, and the patient was quite well and able to work hard in December, 1906.

(19) FRITZSCHE, reported by Schostak, *Beiträge für klinische Chirurgie*, 1907, lvi, 2, p. 368.—A woman, aged 18, in the autumn of the year 1880

suffered from an attack of inflammation of the cæcum, and from more severe attacks in February and July of the following year. In the year 1897 the patient was admitted into hospital with a tympanitic abscess in the right mesogastrium, on opening which fæces escaped; a fæcal fistula resulted. During 1897-8 several unsuccessful attempts were made to close the fistula by suture. In 1899 the abdomen was opened with a view to closing the fistula. A small duodenal fistula was discovered in addition to the cæcal fistula. The operation had to be abandoned on account of adhesions and the friability of the intestine. The patient left the hospital with the fistula smaller, but with constant pain. In September, 1899, the fistulous opening was as big as ever. Anterior gastrojejunostomy was performed in order to divert the gastric contents from the duodenum with a view to a further attempt to close the opening in the duodenum. In February, 1900, there was still a constant flow of intestinal contents from the duodenal fistula; the pylorus was divided and the ends of the stomach and duodenum were closed by suture. In March, 1900, the fistulous opening in the duodenum was closed by suture. In November, 1900, the large fistula in the ascending colon was closed by suture, after destruction of the spur by means of a clamp. Meantime the patient had been suffering from severe pain in the region of the stomach, so severe that morphia was given for its relief. To the left of the navel was a hard induration adherent to the abdominal wall. In July, 1901, the indurated area of the abdominal wall was excised, together with a portion of the jejunum and stomach at the site of anastomosis. The efferent limb of the divided jejunum was implanted in the stomach, and the afferent limb was anastomosed to the efferent (Roux's operation). The patient experienced immediate relief, but shortly after her return home the pain recurred so that she again had recourse to morphia. In June, 1902, a fresh peptic ulcer was excised from the site of anastomosis. The opening in the stomach was closed, and the jejunum reimplanted at another spot. The pain disappeared and the patient returned home, but once again the pain returned, and again she took to morphia. After a time she gradually improved, and two years later was in better health than for many years.

(20) GREGORY CONNELL, *Surgery, Gynæcology, and Obstetrics*, January, 1908, p. 39.—Anterior gastrojejunostomy was performed by Dr. Oviatt, in 1903, on a patient suffering from gastric ulcer with pyloric stenosis and dilated stomach. The operation was followed by relief for two years, after which there was a gradual return and increase of the stomach symptoms. Three years after the operation Dr. Connell reopened the abdomen and found a cicatricial stenosis of the pylorus with dilatation of the stomach, and practical obliteration of the gastro-intestinal anastomosis. At one end of this cicatricial depression, the end towards the cardia, there was marked induration or thickening of the jejunal wall. This mass was about $\frac{1}{2}$ in. in length, $\frac{1}{4}$ in. wide, and elevated $\frac{1}{8}$ in. above the surrounding mucosa. It was fibrous in consistency, with its surface white and smooth. It extended through the jejunal wall to the greater curvature of the stomach, to which it was adherent. A second anterior gastrojejunostomy was made with prompt and thus far (nine months) permanent recovery.

(21) CACKOVIC, *Liecnicki viestnik*, No. 7, and private communication.—Anterior gastrojejunostomy was performed on a man, aged 30. Eight months after the operation symptoms of ulcer appeared. Five months later the abdomen was reopened and a perforated ulcer directly opposite the anastomosis was sutured. The patient recovered. The after-history is unknown.

(22) BERG, reported by Einar Key, *Nordiskt Medicinskt Arkiv*, 1907, xl, afd. 1, heft 2.—Anterior gastrojejunostomy (fundosa) was performed in January, 1896, on a man aged 39. For a short period after the operation the patient remained quite well, and put on 2 st. in weight. During the summer of 1896 and during the ensuing winter he suffered from several attacks of sharp, cutting pains in the abdomen, which he attributed to indiscretions in diet. Between the attacks he was quite well and able to work. In the summer of 1898 he strained himself lifting a heavy weight, and felt a sharp tearing pain in the region of the umbilicus. In December, 1898, he noticed an abdominal lump to the right of the umbilicus, in which he felt pain after taking food in larger quantity than usual. The pain was immediately followed by vomiting, after which the pain disappeared. June, 1898: Readmitted to hospital; total acidity 60. Abdomen reopened. Many adhesions between stomach, liver and neighbouring organs. The abdominal lump was the result of adhesions between the gastrojejunostomy and the anterior abdominal wall. In separating these adhesions the intestine was opened. The aperture was closed by three rows of sutures. The patient recovered, and for a time his condition remained comparatively good. In the spring of 1902 he became worse, vomiting (3 to 4 quarts) became frequent and was independent of the nature of food. Readmitted July, 1902; July 4, abdomen reopened. Many strong adhesions separated and site of gastrojejunostomy exposed. The afferent jejunal loop was greatly hypertrophied, and exhibited strong peristaltic contractions. The efferent limb was small and thin. An entero-anastomosis was performed between the afferent and efferent limbs. He left the hospital somewhat improved on July 26, but subsequent to this date was never really well. Vomiting recurred and necessitated stomach lavage (in 1905 twice daily). In February, 1905, he was again readmitted. There was an ill-defined tender mass around and to the right of the umbilicus; total acidity 50. The abdomen was again opened. After separating many adhesions a perforated jejunal ulcer, adherent to the anterior abdominal wall, was discovered 2 to 3 cm. below the gastrojejunostomy. The portion of the intestine involved was resected. November, 1906: Since the last operation patient has suffered almost daily from colicky pains arising independently of the ingestion of food. To the right of and below the umbilicus is a tender induration in the right rectus muscle.

(23) Operation for jejunal ulcer by BERG (gastrojejunostomy previously by another surgeon), reported by Einar Key (*loc. cit.*).—Anterior gastrojejunostomy was performed in May, 1895, on a woman, aged 38, suffering from pyloric stenosis. In the summer of 1895 she suffered from attacks of pain in the

right inguinal region. In September the pain became constant and radiated upwards, and she also noticed a lump in left side just below the thoracic margin. January 1898: An irregular, hard, tender fixed swelling felt below the left margin of the thorax, just external to the parasternal line. On inflation stomach found to be 6 cm. below the umbilicus. Operation by Professor Berg, January 23. Stomach adherent to abdominal wall in front of swelling on left side. On separating the adhesions a cavity was found extending about 1 cm. into the abdominal wall and communicating with the stomach. The stomach could not be freed entirely without widely opening the intestine at the site of the gastrojejunostomy. The portion of jejunum which had been attached to the stomach (about 10 cm. in length) was dissected off and resected. The efferent limb of jejunum was then implanted into the stomach at the site of the previous gastrojejunostomy, and the afferent limb was implanted into the efferent limb lower down (Roux's operation). The part removed from the stomach and abdominal wall presented at its central point a round cavity, about 1 cm. in depth. Lying free in its most dependent part was a coil of intestinal silk suture about $1\frac{1}{2}$ cm. in length, the ends of which entered the walls of the stomach in the direction of the site of the gastrojejunostomy. The patient remained fairly well until 1900, when she began to suffer again from severe pain, and noticed a swelling in the abdomen. On July 9 the abdomen was reopened. The site of the gastrojejunostomy was adherent to the anterior abdominal wall. On separating the adhesions a cavity was opened, which communicated with the stomach and intestine. Here and there in the walls of this cavity were the ends of sutures. The openings into the stomach and intestine were closed by suture and covered with an omental graft. The raw surface on anterior abdominal wall was similarly covered. The patient died on July 23. Post mortem: Acute peritonitis, perforation of ulcer on the posterior wall of pyloric end of stomach. Several small ulcers existed in the stomach and an ulcer at the site of implantation of the intestine to the stomach.

(24) Reported by Einar Key (*loc. cit.*).—Anterior gastrojejunostomy was performed in 1899 on a man, aged 26, suffering from stenosis of the pylorus and dilatation of the stomach. The patient felt quite well until August, 1903, when, following a movement involving great strain on the abdominal muscles, he again felt diffuse pain and tenderness in the epigastrium. A few days later he became acutely ill with epigastric pain and tenderness below the costal margin to the left of the middle line. After a few weeks he recovered and remained quite well until May, 1904, when on lifting a heavy weight pain in the epigastrium recurred. In June, 1904, a resistant mass was felt in the epigastrium to the left of the middle line; he improved, and the mass could no longer be felt. On returning to work the pain recurred and the swelling was again felt. He was admitted into Professor Berg's clinic in September, 1904. Above the umbilicus and to the left of the middle line a resistant mass was felt, which moved with the abdominal wall. Test meal: Total acidity 87, free hydrochloric acid, bile present. October 10, test meal: Total acidity 29, bile

present. Operation, October 11: The site of the gastrojejunostomy was adherent to the anterior abdominal wall. On separating the dense adhesions a perforated ulcer was found in the jejunum, just below the gastrojejunostomy opening; the opening was sutured and covered with an omental graft. In January, 1906, the patient was very well, and could digest any food taken in moderation; on a few occasions he had vomited after eating or drinking great quantities.

(25) BERG, reported by Einar Key (loc. cit.).—On September 26, 1905, posterior gastrojejunostomy was performed on a woman, aged 26, who had a perforated duodenal ulcer; the ulcer was sutured and the pylorus excluded. Early in November the pain recurred, and on two occasions was accompanied by cramp in the arms and legs. On November 13 the abdomen was reopened; a peptic jejunal ulcer was found in the afferent jejunal limb, $\frac{1}{2}$ cm. from the gastrojejunostomy; the ulcer was surrounded by a mass of adhesions, which had involved and caused a constriction in the efferent jejunal limb. A new gastrojejunostomy was performed (Roux's operation). After the operation the patient continued to vomit daily; on December 3 the total acidity of the vomit was 112. Tetany set in on December 4 and the patient died on the following day. Post mortem: Just below the gastrojejunostomy opening was an ulcer on the posterior wall of the jejunum, about the size of a penny; tuberculosis of some of the mesenteric glands.

(26) ROTGANS, *Congrès de la Société internationale de Chirurgie*, Brussels, 1905, p. 170.—This surgeon, at the first congress of the International Society of Surgeons, mentioned the case of a man from whom he excised a peptic jejunal ulcer following anterior gastrojejunostomy; the patient recovered.

(27) CACKOVIC, *Liecnicki viestnik*, 1903, No. 7, and private communication.—Anterior gastrojejunostomy was performed on a man aged 36. Two days after the operation the patient vomited blood; eight days after the operation the vomit became bilious in character, and on the sixteenth day the abdomen was reopened; after separating adhesions a perforated ulcer on the efferent jejunal limb was discovered and closed by suture, and an entero-anastomosis between the afferent and efferent jejunal limbs was performed. Three months later a fistula formed, which under medical treatment gradually healed, so that the patient left the hospital seven months after the original operation. Twenty-two months later a fistula again formed, but healed after three months. The later history of the patient is unknown.

(28) PATERSON (*see full notes at commencement of paper, p. 238*).

(B) CASES IN WHICH THE ULCER BECAME ADHERENT TO AND
PERFORATED INTO THE COLON.

(29) KAUFMANN, *Mitteilungen aus den Grenzgebieten der Medizin und Chirurgie*, Jena, 1905, xv, p. 151.—In June, 1901, anterior gastrojejunostomy with entero-anastomosis was performed on a man, aged 41, who had

suffered from gastric trouble for ten years. The patient gained 20 lb. in weight and remained in good health for three months. Then his symptoms recurred, feeling of weight and pain some hours after food. In May, 1902, examination showed that the stomach was dilated and marked hyperacidity was present. In April, 1903, severe abdominal pain necessitating the use of morphia. In November, 1903, eructations having an odour of sulphuretted hydrogen, followed in a few days later by vomiting of faecal material. A diagnosis of gastro-colic fistula was confirmed by the injection of gentian violet into the rectum. The fluid injected was drawn off from the stomach on passing a stomach tube. The patient's condition gradually becoming worse, the abdomen was reopened in March, 1905. The transverse colon was extensively adherent to the stomach and jejunal loop, and there were two fistulous openings, one between the transverse colon and stomach, and the other between the transverse colon and the jejunal loop. After separation of the adhesions the openings were closed by suture. The calibre of the colon was so narrowed by the adhesions and by the suturing that it was deemed advisable to perform an anastomosis between the transverse colon and the sigmoid flexure. This was effected by means of a Murphy's button. Six days later the patient died from perforation of the sigmoid caused by sloughing of the intestine round the button. At the post mortem it was seen that the fistula between the colon and jejunum was in the efferent jejunal limb. The afferent limb was very short. The gastrojejuno-stomy opening had become completely obliterated.

(30) CZERNY, *Beiträge zur klinische Chirurgie*, xxxix, p. 99, and private communication. — In November, 1900, Czerny performed posterior gastro-jejuno-stomy, by means of a Murphy's button, on a man aged 36, suffering from ulcer of the stomach accompanied by dilatation and hyperacidity. The patient remained well until June, 1902, at which time he began to suffer pain. The presence of faecal material in the stomach was noted. In November, 1902, the abdomen was reopened. On the level of the anastomosis a peptic ulcer was discovered extending towards the anterior stomach wall. The ulcer was adherent to the transverse colon into which it had perforated. The hole in the transverse colon was closed and a new gastrojejuno-stomy with a Murphy's button was performed at another part of the stomach. The patient recovered.

Professor Czerny has been good enough to inform me that this patient is at the present time alive and well and working as a gardener.

(31) GOSSET, *Revue de Chirurgie*, 1906, xxxiii, p. 59, and private communication. — Posterior gastrojejuno-stomy was performed in December, 1903, on a man, aged 40, suffering from the effects of pyloric stenosis. He remained well until July, 1905, gaining 50 lb. in weight. In July, 1905, he slipped and fell in the street, an accident which was immediately followed by a recurrence of pain in the left side. He began to get thin, and vomiting recurred. The vomit was bilious in character with a faecal odour. The patient also had persistent diarrhoea, and between July and September lost 22 lb. in weight. At the end of September, 1905, the abdomen was reopened. The efferent

jejunal limb was considerably dilated, and 4 in. below the anastomosis it was adherent to the posterior surface of the transverse colon. On liberating these viscera a large communication was found to exist between the jejunum and transverse colon. The two apertures were closed by suture, and as the transverse colon was somewhat constricted by this procedure, an ileo-sigmoidostomy was performed. The patient made an uninterrupted recovery.

Dr. Gosset informs me that at the present time (April, 1908) this patient is in perfect health.

(32) HERCZEL, Congress of French Surgeons, 1905.—In this case, following a posterior gastrojejunostomy performed for pyloric stenosis, a peptic jejunal ulcer formed in the efferent jejunal loop, became adherent to, and perforated into the colon, thus producing a jejuno-colic fistula.

(33) CACKOVIC, *Liecnicki viestnik*, 1909, No. 5.—Posterior gastrojejunostomy was performed on a man, aged 38, who had suffered from gastric trouble since the age of 16. Three years and two months after gastrojejunostomy symptoms of ulcer were noted. No further operation was performed, and the patient died six years after the gastrojejunostomy had been performed. At the post-mortem an ulcer of the jejunum was found opposite the anastomosis. The ulcer had perforated through the posterior wall of the colon.

Doubtful Cases.

(1A) FRAENKEL, *Deutsches Archiv für klinische Medizin*, 1905, lxxiv, p. 217.—Anterior gastrojejunostomy with entero-anastomosis was performed on a patient, whose sex and age is not stated, in January, 1904. On December 6 of the same year the patient was seized with sudden pain in the side, and a resistance could be felt to the left of the middle line below the navel. An abscess was opened, and later bowel contents flowed from the abscess cavity. By January 10 of the next year the fistula was closed.

(2A) TIEGEL, *Mitteilungen aus den Grenzgebieten der Medizin und Chirurgie*, xiii, 1904, p. 909.—In June, 1901, an anterior gastrojejunostomy with entero-anastomosis was performed on a man, aged 41, who suffered from symptoms of pyloric stenosis. He remained well until August, 1903, when he was seized suddenly with severe pain, below the right costal arch. The pain, which was independent of food, came on at intervals of several days, or sometimes of weeks. On examination there was a diffuse resistance tender to the least touch; 25 c.c. of liquid, without food remains, were drawn off from the fasting stomach; total acidity 28, free hydrochloric acid 12. Three-quarters of an hour after a test breakfast of oatmeal soup, 140 c.c. drawn off; total acidity 49, free hydrochloric acid 23. Five hours and a half after a test dinner, 130 c.c. drawn off; total acidity 34, free hydrochloric acid 9. Under treatment the patient improved, and remained well until March, 1904, when the attacks of pain recurred.

(3A and 4A) HAHN, quoted by Gosset, *Revue de Chirurgie*, 1906, xxxiii, p. 302.—At the German Congress in 1902 Hahn mentioned two cases in which after gastrojejunostomy for non-malignant disease, in which ulcers had perforated and resulted in abscesses of the abdominal wall, both patients recovered. Further details are not available.

(5A) CACKOVIC, *Liecnicki viestnik*, 1903, No. 7, and private communication.—Anterior gastrojejunostomy was performed on a woman aged 40. Six days after the operation she suffered from spasmodic pain, and later she had a painful swelling of the anterior abdominal wall. Under anti-acid treatment for some months the patient recovered, and seventeen months later was "entirely sound" (letter from Dr. Cackovic).

(6A) LYLE, *New York Medical Journal*, 1906, lxxxiv, p. 1230.—The abdomen of a man, aged 26, was opened on account of sudden perforation of gastric ulcer near the pylorus. The perforation was closed by suture and posterior gastrojejunostomy with entero-anastomosis was performed. Six months later symptoms of ulcer returned which were relieved by medical treatment.

(7A) SCHOSTAK (loc. cit.).—In March, 1901, anterior gastrojejunostomy was performed on a man, aged 70, who had for several years suffered from symptoms of pyloric obstruction. In March, 1902, pain recurred, at first in the region of the liver, so that a suspicion of gall-stones was raised. In July a hard, tender induration was felt to the left of the operation scar, adherent to the abdominal wall. Under medical treatment he gradually improved and the swelling disappeared. In the autumn of 1902 he still remained well.

(8A) HADRA, *Kongressbericht*, 1900, p. 152.—Anterior gastrojejunostomy was performed in May, 1899, on a young man suffering from pyloric stenosis. He remained well for six months, and then had recurrence of pain. Between the ensiform cartilage and the navel was an infiltration the size of the hand. Incision of a small blister opened a gastric fistula. The fistula closed, but the pain continued. With absolute rest and administration of alkalis the pain disappeared. The case was considered to be one of peptic ulcer.

(9A) PATERSON (see report at p. 245).

(10A) NYROP, *Ugeskrift für Laege*, 1907, p. 54.—Einar Key mentions a case of supposed jejunal ulcer recorded by Nyrop. I have not been able to obtain a copy of the original paper, so cannot give further details.

The Sex of the Patients.

A striking feature with regard to the recorded cases is the large proportion of male patients. Disregarding the doubtful cases, the sex of the patient is mentioned in fifty of the fifty-two cases here presented, and of these, thirty-nine were men and only eleven women, a proportion of 78 per cent. of men. The reason for this disparity in the incidence

of jejunal ulcer in the two sexes is not clear. Schostak suggests that the men may not pay sufficient regard to the directions given to them as to diet after operation, and that their predilection for alcohol may act as a predisposing cause. Both these factors may play a part; in one instance the patient was known to be a heavy drinker, and perforation occurred while he was drunk (Group I., Case No. 10), but they will hardly account for such a marked disproportion. I think that possibly a reason is to be found in the fact that a greater number of men than women are treated surgically for gastric and duodenal ulcers. Dr. W. J. Mayo [11] reports that of his last 100 operations for gastric or duodenal ulcer, sixty-two of the patients were men and thirty-eight females.

The Age of the Patients.

The majority of the patients belong to the middle period of life. The youngest was a female infant, aged two months, on whom gastrojejunostomy was performed for infantile pyloric stenosis. The oldest patient was aged 58, although among the cases classified as doubtful is that of my patient, aged 76. The ages of the patients in those cases in which the age is stated are shown in the following tables:—

TABLE I.—AGES OF MALE PATIENTS.

Aged 20 to 29	8 patients
" 30 " 39	12 "
" 40 " 49	10 "
" 50 " 60	4 "

TABLE II.—AGES OF FEMALE PATIENTS.

Aged under 20	2 patients
" 20 to 29	3 "
" 30 " 39	3 "
" 40 " 50	2 "

TABLE III.—AGES OF MALE AND FEMALE PATIENTS.

Aged under 20	2 patients
" 20 to 29	11 "
" 30 " 39	15 "
" 40 " 49	12 "
" 50 " 60	4 "
Age not stated	8 "
				52

Average age: Men, 37.6 years; women, 31.3 years.

The Condition for which the Original Gastrojejunostomy was performed is shown in the following Table:—

TABLE IV.—CONDITIONS FOR WHICH GASTROJEJUNOSTOMY WAS PERFORMED.

Pyloric stenosis	33 cases
Dilated stomach	2 "
Gastric ulcer	2 "
Gastric trouble	6 "
Duodenal fistula	1 case
Cancer of stomach	1 "
Not stated	7 cases
				—
				52

The Type of Gastrojejunostomy performed is shown in the following Table:—

TABLE V.—TYPE OF GASTROJEJUNOSTOMY PERFORMED.

	Group I	Group II	Totals
Anterior	11	16	27
Anterior, with entero-anastomosis	2	8	10
"En—Y"	1	1	2
Posterior	4	8	12
Supracolic	1	0	1

The Duration of the Interval between Gastrojejunostomy and the Onset of Jejunal and Gastrojejunal Ulcer.

Jejunal ulcer, like gastric ulcer, may run a perfectly symptomless course until the onset of sudden perforation. This was the case in the majority of cases in Group I. In the cases of Group II, it is probable that the ulcer had existed for a considerable time before the onset of symptoms. According to Lennander [10], the reason for this absence of pain in the early stage of these ulcers is that there are no pain-perceiving nerves in the stomach and intestine. Ulceration of these viscera, therefore, is painless until it invades the peritoneum. We have thus no means of determining the time of onset of ulceration; the nearest we can get is to take the time of the onset of the first symptoms. In the recorded cases the shortest interval was two days (Group I, No. 18), and the longest interval eight years (Group II, No. 7).

The varying periods between the original gastrojejunostomy and the onset of symptoms are shown in the following table:—

TABLE VI.—INTERVAL BETWEEN GASTROJEJUNOSTOMY AND ONSET OF SYMPTOMS.

Under 1 month	5 cases
2 to 3 months	6 "
3 to 6	7 "
6 months to 1 year	11 "
1 to 2 years	8 "
2 to 3	4 "
Over 3 years	8 "
Interval not stated	3 "
				52

We see thus that in 56 per cent. of the cases in which data are available the symptoms of ulcer appeared within a year, and in 73 per cent. within two years, of gastrojejunostomy. The average of all the cases is twenty months.

The Mortality from Jejunal and Gastrojejunal Ulcer following Gastrojejunostomy.

The immediate results in the cases of this series are shown in the following tables:—

TABLE VII.—MORTALITY—ALL CASES (GROUPS I AND II).

Recovered	29
Died	22
Result not stated	1
					52

TABLE VIII.—MORTALITY—UNRECOGNIZED OR UNTREATED CASES (GROUP I).

Recovered	0
Died	13
					13

TABLE IX.—SHOWING MORTALITY IN CASES TREATED BY OPERATION (GROUPS I AND II).

Recovered	29
Recovered after first but died after subsequent operation	2
Died after first operation	6
Result not stated	1
						38

Thus the rate of immediate mortality in all the cases of the series, whether an operation was performed or not, is 42 per cent. Of the cases

treated by operation the mortality rate is 15 per cent., but two of the patients who recovered from a first operation for jejunal ulcer succumbed to a second or subsequent operation. If these two fatalities are included, then the ultimate mortality rate of the operation cases is 21 per cent.

It is noteworthy that the death-rate in the cases of Group I treated by operation is identical with the death-rate of the cases in Group II (16 per cent.) ; indeed, if the cases in Group II which proved fatal after a second or subsequent surgical intervention are included, the death-rate of the cases in Group II is actually higher than that of the cases in Group I. In other words, the cases in which general peritonitis ensues are less fatal than those cases in which general peritonitis is prevented by the formation of protective adhesions. This is certainly not what at first sight we should expect. Although the cases as yet recorded are too few in number to warrant us in assuming that this is the rule, the higher mortality rate of the cases in Group II is not surprising when we consider that the existence of extensive adhesions, in addition to the occasional necessity of intestinal resection, renders operation undertaken in this group of cases a very formidable procedure, as a perusal of the recorded cases will convince those who have had the good fortune not to have had to deal with this complication.

The Influence of the Type of Gastrojejunostomy on the Mortality.

Of the thirty-nine cases in which the anterior type of operation was performed, perforation into the general peritoneal cavity occurred in 14 per cent. or 35 per cent. ; while of the twelve cases in which the posterior operation was performed, perforation ensued in 34 per cent. Whether these figures represent the relative risks of perforation into the general peritoneal cavity in the two groups, it is impossible to say, having regard to the small number of cases. It would appear, however, that the mortality, after operation in cases in which the posterior operation has been performed, is considerably higher than in those in which the anterior has been performed. Including all the cases, whether an operation was performed or not, the comparative mortality rate is as follows :—

TABLE X.—COMPARATIVE MORTALITY RATE—ALL CASES.

		Recovered		Died		Mortality rate
Anterior operation	...	26	...	13	...	33 per cent.
Posterior ,,	...	3	...	8	...	72 ,, .

If, however, we do not take into account those cases in which no operation was performed, twelve in number (one fatal supracolic operation is not included in these figures), the difference is less, but still very marked:—

TABLE XI.—COMPARATIVE MORTALITY RATE—OPERATION CASES.

		Recovered		Died		Mortality rate
Anterior operation	...	26	...	5	...	16 per cent.
Posterior "	...	3	...	3	...	50 "

This difference is so striking that we can hardly doubt that it is one of cause and effect, and it is what *a priori* we should expect, having regard to the different conditions obtaining after the two types of gastrojejunostomy. In jejunal and gastrojejunal ulcer following the anterior operation, the affected portion of intestine is near the surface; indeed, in eighteen of the twenty-five anterior operation cases in Group II the ulcerated portion of intestine was adherent to the abdominal wall. After the posterior operation, on the other hand, the ulcer is very deeply situated, and adhesions to the omentum and neighbouring viscera may render access to it very tedious and difficult.

The Symptomatology of Jejunal and Gastrojejunal Ulcer.

The clinical picture presented by the cases in which perforation into the general peritoneal cavity occurs differs materially from that in cases in which by the formation of protective adhesions general peritonitis is prevented.

GROUP I.

In almost all the cases comprised in this group the patients had after gastrojejunostomy lost all gastric symptoms and had been in good health until suddenly, "like a bolt from the blue," as Schostak graphically puts it, they were seized with sudden severe pain, heralding a perforation peritonitis which in the untreated cases speedily had a fatal termination. In four only of the cases in this group were there any previous indications that the result of the gastrojejunostomy had not been entirely satisfactory. In one case (No. 1, Group I), the patient had a recurrence of his symptoms soon after leaving the hospital, and, on readmission, examination showed that the stomach was dilated below the umbilicus; under treatment he improved, and remained well until the sudden seizure some months later. In another case (No. 8), two weeks

before death vomiting and melæna occurred. In case No. 15 vomiting recurred five months before death, and in one case (No. 19) there is a history of a sudden attack of pain two years previous to the attack which proved fatal. It is probable that an ulcer existed at the time of the first attack, but that general peritonitis was prevented at that time by the formation of adhesions, a conjecture supported by the discovery at the post mortem of a mass of adhesions round the afferent limb of the jejunum. In one case (Group I, No. 3) peritonitis was the result, not of perforation of an ulcer, but of a subphrenic abscess secondary to a jejunal ulcer which perforated all the intestinal coats except the serosa. In two instances (Group I, cases No. 2 and 11) the seizure followed immediately on the lifting of a weight. Probably in all these cases there were slender protective adhesions the separation of which resulted in the escape of bowel contents. Mr. Battle's case is probably unique, for this patient was operated on for perforation into the general peritoneal cavity on three occasions—once for perforation of a gastric and twice for perforation of a jejunal ulcer—with a successful result on each occasion.

GROUP II.

Pain varying in intensity, and in many of the cases of a severe character, is the prominent symptom of the cases in this group. In some instances the pain was not sufficiently severe to prevent the patients from following their usual vocations, more or less, for some years before they sought surgical relief, while in others the patients were reduced to an extreme degree of exhaustion. The pain does not appear to have any definite relation to the ingestion of food. In three of the cases (Group II, Nos. 3, 11 and 14) this is specifically mentioned. In five instances the pain was aggravated by food—in three cases immediately (Group II, Nos. 9, 22 and 28), and in two cases (Group II, Nos. 4 and 6) one to two hours later. Vomiting is noted as having occurred in four instances (Group II, Nos. 2, 3, 10, and 22). I think that vomiting in these cases may be regarded as evidence of obstruction. Whether the obstruction is antecedent to or consequent on jejunal ulcer is a question which I will discuss further on. In one case (Group II, No. 10) the vomit was chiefly blood, and in one case (Group II, No. 3) there was melæna as well as vomiting.

An important sign of jejunal or gastrojejunal ulcer is the existence of an induration or swelling in the abdominal wall. This condition was noted in nineteen of the twenty-eight patients in Group II. The

swelling is the result of the adhesion of the ulcerated area to the anterior abdominal wall; perforation of the base of the ulcer leads to an inflammatory exudation into the abdominal wall and consequent induration, and in some instances to a very definite mass. In one of my own cases (Group II, No. 28) the skin over the swelling became ulcerated and destroyed, so that a jejunal fistula formed. The situation of the swelling is usually in the left rectus muscle about the level of the umbilicus (twelve cases). In four cases the swelling was in the right rectus, and in two in the middle line. In all of these cases the anterior operation had been performed. In the four cases of this group in which posterior gastrojejunostomy had been performed no mention is made of the existence of a swelling, but in one case (Group II, No. 7) it is stated that there was a resistance in the middle line. The fixing of the jejunal loop in front of the colon in the anterior operation readily explains the frequency of a palpable swelling in these cases. The presence of a swelling in the left rectus, then, is highly suggestive, if not indeed diagnostic, of gastrojejunal or jejunal ulcer, while the absence of any induration or swelling renders the diagnosis of this condition more difficult and less certain. In the cases in the second division of Group II signs of a gastrocolic or jejunocolic fistula were present. Thus, in case No. 29 the patient vomited faecal material, in No. 30 faecal material was found in the stomach on passing a stomach tube, while in No. 31 the patient vomited a bilious material with a faecal odour. In these three patients pain was a prominent symptom. Of case No. 32 I have been unable to obtain further details.

The Condition of the Gastric Contents.

For obvious reasons gastric analysis was impossible in the cases belonging to Group I. Even in the cases of Group II the references to the condition of the gastric contents are scanty. From the evidence of the figures given my impression is that such examinations as were made were done by Töpfer's method, which, as I have already pointed out, is unreliable. My own case appears to be the first case of jejunal ulcer in which any attempt has been made accurately to analyze the gastric contents, and I have to regret that an accurate gastric analysis was not made at the time of the original gastrojejunostomy. Some information as to the gastric contents after gastrojejunostomy is given in eighteen of the cases of Group II, and in five of them the amount

of free hydrochloric acid is stated. Classifying these eighteen cases, we find :—

TABLE XII.—THE ACIDITY OF THE GASTRIC CONTENTS IN JEJUNAL AND GASTRO-JEJUNAL ULCER.

High total acidity	9 cases
Normal total acidity, with excess of free hydrochloric acid	4 „
Normal or sub-normal acidity	5 „
					<hr/> 18

From these figures we may conclude that hyperacidity after gastro-jejunosomy was present in thirteen of the eighteen cases in which the gastric acidity was investigated.

The Diagnosis of Jejunal and Gastrojejunal Ulcer.

In the cases in which perforation into the general peritoneal cavity occurs the signs and symptoms are similar to those of a perforated gastric ulcer. We cannot distinguish between the two conditions, but it appears that after gastrojejunosomy a perforated jejunal ulcer is a more likely event than a perforated gastric ulcer.

The diagnosis of jejunal or gastrojejunal ulcer when protective adhesions are present must often be uncertain. But this much we can say : that when, after an interval of good health, a patient who has had gastro-jejunosomy performed begins to suffer constant pain, especially if the pain is accompanied by hyperacidity or hypersecretion, we should bear in mind the possibility of the existence of a jejunal or gastrojejunal ulcer. When in addition to pain there is an induration or swelling of the abdominal wall in the neighbourhood of the stomach the diagnosis is almost certain. We must remember, however, that a swelling or induration is rarely present in patients on whom the posterior operation has been performed. For practical purposes we may say that every case of recrudescence of pain of a constant character after gastrojejunosomy should be regarded as a case of potential jejunal or gastrojejunal ulcer, and treated accordingly.

The Pathology of Jejunal and Gastrojejunal Ulcer.

In nearly one-third of the recorded cases of jejunal ulcer the ulcer was probably gastric rather than jejunal. By this seeming paradox I mean that under the term "jejunal ulcer" all writers hitherto have included

the cases in which the ulceration is at the site of the anastomosis. Now, in these cases it is apparently the gastric mucous membrane that is chiefly involved, and in all probability such ulcers originate in the gastric mucous membrane surrounding the anastomotic opening. I think a distinction should be made between these ulcers and the ulcers which occur in the jejunum. Their characters differ, and I believe the conditions determining their occurrence are dissimilar. For the ulcers at the site of the anastomosis I would suggest the term "gastrojejunal." The situation of the ulcers in the cases under consideration is shown in the following table:—

TABLE XIII.—SITE OF ULCER IN RECORDED CASES.

Jejunal:				
Afferent limb	2 cases
Efferent "	12 "
Opposite anastomosis	5 "
Limb not stated	13 "
Gastrojejunal	14 "
Multiple ulcers	6 "
				—
				52

As regards the cases in which there was more than one ulcer. In one case (Group I, No. 4) there were four ulcers, two in the efferent and two in the afferent limb; in one case (Group I, No. 8) there was a perforated ulcer in the afferent limb, and several other ulcers in the neighbourhood of the anastomosis; in one case (Group I, No. 17) the ulcers were in the afferent limb; in one case (Group I, No. 18) there were four perforated ulcers in the efferent limb; and in two cases (Group II, Nos. 13 and 29) there was an ulcer at the site of the anastomosis as well as one in the jejunum.

First, as to jejunal ulcer proper. This in appearance and clinical course bears a close resemblance to the ordinary round ulcer of the stomach and duodenum. As in the stomach, we have the "punched-out" ulcer suggestive of rapid destruction of tissue, apt to perforate before protective adhesions are formed. Naturally, the thin jejunal wall is more readily destroyed by ulceration than is the gastric wall, with its thicker musculature and tough submucous layer. Then, too, we have the jejunal ulcer with shelving thickened edges, more chronic in course, becoming adherent to neighbouring structures, finally perforating and leading to inflammatory infiltration of the adherent structures. Jejunal ulcers are most commonly situated in the efferent limb, either opposite to or just below the anastomosis, an observation which, as I shall point out later, has a bearing on the ætiology of these ulcers.

Secondly, as to gastrojejunal ulcer (ulcer at the site of anastomosis). Gastrojejunal ulcer lacks the definite localized appearance of the jejunal ulcer. It appears as an irregular ulceration round the margin of the anastomotic opening, sometimes involving the jejunal as well as the gastric mucosa. If healing occur, the process of cicatrization may lead to partial (*see* cases Group I, No. 15, and Group II, No. 20) or even complete (*see* cases Group II, Nos. 6 and 29) obliteration of the communication between the stomach and jejunum. To this point I shall refer later. Gastrojejunal ulcers are less likely than jejunal ulcers to perforate into the general peritoneal cavity; for of the fourteen recorded cases of gastrojejunal ulcer, perforation into the general peritoneal cavity occurred in three (21 per cent.), while of the thirty-five cases of jejunal ulcer, general peritonitis ensued in sixteen (45 per cent.).

The *Æ*tiology of Jejunal Ulcer.

Various hypotheses have been adduced as furnishing the explanation of jejunal ulceration following gastrojejunostomy; and, although until our knowledge as to the causation of gastric and duodenal ulcers is more precise, conclusions regarding the *æ*tiology of jejunal ulcer may require subsequent modification, nevertheless we can form fairly reliable indications as to the conditions favouring its occurrence, and so come to some conclusions as to the best means of prevention and treatment. The three chief hypotheses which have been put forward to explain the occurrence of jejunal ulcers are as follows:—

(1) That Jejunal Ulceration is due to Circulatory Disturbances in the Attached Jejunum.

Tiegel [22] suggests that the circulation of the jejunum may be interfered with in several ways: (a) The loop of jejunum which passes in front of the transverse colon may be of insufficient length, and so may be subject to tension, or the blood-supply may be impeded by kinks in the mesentery itself; (b) arterio-sclerosis as in Steinthal's case (Group I, No. 4), in which atheroma was associated with a slight kinking of the mesentery; (c) injury to the mucous membrane, either at the time of operation, or later by hard particles of food. A. H. Gould further suggests that circulatory disturbances are more likely to occur when the jejunum is attached by the anterior than by the posterior method.

(2) That Jejunal Ulcer is an Infective Process.

The hypothesis that jejunal as well as gastric ulcers are of infective origin has been advocated by Neumann [12]. In support of his view he calls attention to the frequent multiplicity of such ulcers, and with regard to jejunal ulcer he points out its progressive character and tendency to invade neighbouring tissues, and contrasts it with the limited necrosis produced by circulatory disturbance, and he maintains that ulceration presenting this progressive character is invariably of infective origin.

(3) That Jejunal Ulcer is due to Hyperacidity of the Gastric Contents (Peptic Hypothesis).

After gastrojejunostomy the gastric contents escape directly into the jejunum without the previous modification which results, under normal conditions, from their mixture with the alkaline bile and pancreatic secretion in the duodenum. Jejunal ulcer, according to the peptic hypothesis, is due to the digestive action of hyperacid gastric juice on a mucous membrane accustomed only to the presence of alkaline contents.

No single one of these hypotheses affords a satisfactory explanation of all the cases so far observed. The evidence that circulatory disturbances play a part in the causation of jejunal ulcers is very inconclusive. From experience in abdominal surgery we know that the small intestine may form adhesions, and may be markedly kinked in places without prejudice to its blood-supply. Surely a kink or twist of a degree sufficient to cause interference with the intestinal blood-supply would be obvious at a post-mortem examination, but so far in one case only is there any evidence of circulatory disturbance, and in that case the condition found was a slight mesenteric kink and atheroma of the vessels, which may or may not have favoured the occurrence of the ulcer which existed. The infective hypothesis affords a probable explanation of those cases (Group I, Nos. 4, 8, 12, 17, and 18) in which jejunal ulceration occurred within a very short period of the performance of gastrojejunostomy, and it is significant that in four of these five cases the ulcers were multiple. In contrast to this we find that in none of the cases in which ulceration occurred at a later period after gastrojejunostomy was there more than one ulcer in the jejunum. This fact, the limited area affected, and the occurrence of spontaneous

healing in some instances, are against the general application of the infective hypothesis, and the progressive character of the ulcer in some cases can be explained without supposing an infective origin.

The peptic hypothesis has been adopted by most of those who have written on this subject, so that jejunal ulcer following gastrojejunostomy is usually spoken of as "peptic" jejunal ulcer. This appears to me "putting the cart before the horse"; the digestion of the living cells must be a consequence of their injury by the hydrochloric acid of the gastric juice. The peptic action is secondary, the primary factor being the injury of the mucous membrane by the toxic action of hydrochloric acid. And may I again emphasize my belief that the amount of free hydrochloric acid, not the total acidity of the gastric contents, is the important factor? The free hydrochloric acid acts as a poison on the cells of the mucous membrane, so injuring them that they fall a ready prey to digestive action. In a normal individual, probably all the free hydrochloric acid of the gastric contents is neutralized before the chyme leaves the duodenum. The neutralizing power of the bile and pancreatic juice is well illustrated by the analyses made in the case of my patient. The fluid escaping from the jejunal fistula contained 0.03 per cent. less free hydrochloric acid than the gastric contents, a difference which shows that the bile and pancreatic juice can more than neutralize all the free hydrochloric acid present in normal stomach contents. Now after gastrojejunostomy the gastric contents pass directly into the jejunum. Two circumstances, however, prevent this diversion of the gastric contents from exercising an injurious effect on the jejunal mucous membrane. As I have pointed out elsewhere [13], after gastrojejunostomy there is, first, a distinct diminution of the chlorides secreted by the stomach, and, secondly, some regurgitation of bile and pancreatic juice into the stomach. In consequence of these changes the percentage of free hydrochloric acid in the gastric contents is markedly diminished, so that after a satisfactory gastrojejunostomy the amount of free hydrochloric acid entering the jejunum is so small that it is at once neutralized by the bile and pancreatic juice. This view is supported by observations on my patient. When after a period on milk diet the amount of free hydrochloric acid in the gastric contents had fallen to 0.018 per cent.—a percentage, be it said, considerably greater than is usually present after gastrojejunostomy—the fluid collected from the fistula contained merely a trace of free hydrochloric acid (0.004 per cent.).

As I have already mentioned, either hyperacidity or excess of free

hydrochloric acid was present in thirteen of the eighteen cases in which the state of the gastric contents was noted. Hyperacidity, therefore, is very frequently associated with jejunal ulcer. Is the relation one of cause and effect?

The situation of the ulcers is very significant. In five cases only was there an ulcer in the afferent jejunal limb (Group I, Nos. 4, 8, 17, and Group II, Nos. 2 and 25), and in three of these cases the ulcers were multiple (in cases No. 4 and 8, ulcers in both afferent and efferent limbs), probably indicating an infective origin. Thus, ulceration was limited to the ascending limb in three cases only (Group I, No. 17, and Group II, Nos. 2 and 25). Of these three cases, the first, as I have already indicated, was probably infective; in the second case an entero-anastomosis had been performed, so that the afferent limb was not protected by alkaline bile and pancreatic juice; while that the third case was infective is suggested by the circumstance that the gastro-jejunosomy was performed a few weeks previously for a perforated duodenal ulcer, and that the patient died with tetanic symptoms. We find, then, that of the twenty-three cases in which the exact site of the jejunal ulceration is given, in twenty the ulcer was either opposite the anastomosis or just below it in the efferent limb—that is, just where the acidity of the gastric contents when they reach the jejunum is greatest. These facts appear to me to afford strong evidence in favour of the view I am urging—namely, that jejunal ulcer is the result of the toxic action of free hydrochloric acid on the jejunal mucous membrane. In this connexion an observation of Kocher's [9] is of considerable interest. On reopening the abdomen of a patient whose gastro-jejunosomy opening did not appear to be acting satisfactorily, he found that the obstruction was not at the gastrojejunosomy opening, but immediately below it, in the efferent limb of the jejunum, which was in a state of strong muscular contraction. Kocher is of opinion that the acid gastric juice may stimulate circular contraction of the jejunum just below its junction with the stomach, with the formation of a kind of cul-de-sac in which the stay of the gastric juice may be prolonged and so cause ulceration.

I will at this stage call attention to some other facts which seem to me to warrant the assumption that it is the presence of hydrochloric acid in the jejunum which is the cause of jejunal ulcer. In operating on my own patient I resected a portion of jejunum together with the skin and portion of abdominal wall surrounding the fistula. On examining the specimen I found that the ulcer had completely

healed; the margin of the jejunal mucous membrane was continuous with the scar tissue surrounding the fistula without any sign of ulceration. Why had the ulcer, which must have existed for several months at least, healed in less than four weeks? I think that there is only one satisfactory explanation, and this is, that the healing was the consequence of the diminution, as shown by analysis, of the free hydrochloric acid in the jejunum. Whether the diminution was the result of milk diet or of the rapid escape of intestinal contents which took place through the fistula, is immaterial to the argument. Confirmatory evidence is presented in the records of several other cases. Mikulicz in one of his cases (Group II, No. 2) performed jejunostomy; the patient immediately improved, and three months later the fistula was allowed to close. Shortly afterwards the patient's symptoms returned. Dr. Quénu in his case (Group II, No. 3) resected a portion of jejunum and performed a gastrojejunostomy "En—Y." After a few months the patient had recurrence of his symptoms, and Dr. Quénu then performed a jejunostomy. The patient has remained well ever since (six years), and Dr. Quénu makes the suggestion that the jejunal opening has served as a safety valve to allow of the escape of some of the acid gastric contents. Dr. Brodnitz's patient (Group II, No. 13) was also after a time relieved by a jejunostomy. In one of Dr. Cackovic's patients (Group II, No. 27) a fistula formed, then healed under treatment, and at a later time again formed. Haply the formation of a fistula is Nature's method of getting rid of the excess of hydrochloric acid, and so bringing about a cure. In Dr. Gosset's case (Group II, No. 31) it is interesting to note that the jejunal ulcer perforated into the colon, but at the operation the mucous membrane of the jejunum was continuous with that of the colon without any sign of ulceration. Why did the jejunal ulcer heal? Is not this another instance of the safety valve, the new opening allowing a free escape of the acid juice?

I will now briefly discuss an objection to the view that jejunal ulcer is due to the toxic action of free hydrochloric acid on the jejunal mucous membrane. As I have already pointed out, in some of the recorded cases the acidity of the gastric contents was normal or sub-normal. How are we to account for these cases? It appears to me that those who argue that such cases disprove the hyperacidity hypothesis, overlook several important points. In the first place, let me point out that, although the acidity of the gastric contents may be normal when the patient comes under observation, this does not negative the possibility that at an earlier

stage of the disease hyperacidity may have existed. For example, in my own case, if a gastric analysis had not been made soon after admission there would have been no evidence of the enormous increase of free hydrochloric acid which existed at that time, as at the time of the second analysis the amount of free hydrochloric acid had fallen slightly below normal. This diminution of acidity under dietetic treatment is also illustrated in Cases No. 11 and 24, Group II.

In the second place, although the total acidity of the gastric juice may be normal, the amount of gastric juice secreted may be excessive; in other words, hypersecretion may exist without hyperacidity. If there is hypersecretion, although the percentage of free hydrochloric acid in the gastric juice be normal, the quantity entering the jejunum may be such that it is incompletely neutralized by the bile and pancreatic juice. It is significant that hypersecretion was present in two at least of the cases in which the total acidity was normal (Group II, Cases No. 15 and 22).¹

Thirdly, although the acidity of the gastric contents may be normal, the secretion of alkaline pancreatic juice and bile may be deficient, and so the gastric contents may be imperfectly neutralized in the jejunum. This is, of course, a speculation unsupported by any evidence, but it appears to me a reasonable supposition. If this be the case, then any temporary diminution of the flow of bile or of the secretion of pancreatic juice may favour the occurrence of jejunal ulceration.

Schostak [19] relates a case in which gastrojejunostomy was performed to short-circuit the gastric contents, preparatory to closing a duodenal fistula. Subsequently an operation was necessary on account of a jejunal ulcer (Group II, Case No. 19). Commenting on this case, he says that, as the patient had no gastric symptoms, it may be concluded that the gastric contents were normal and not hyperacid; and he maintains that the case shows that jejunal ulcer may occur with perfectly normal gastric acidity. In my opinion, no such conclusion is warranted. First, the absence of symptoms is no proof that the stomach contents were normal; secondly, in the absence of an analysis, to assume that the gastric contents were normal is unjustifiable; thirdly, from a perusal of the history it appears quite possible that hyperacidity may have existed. I have found that hyperacidity is a frequent concomitant of appendicitis; this patient had suffered from appendicitis, and so I argue

¹ Dr. Cackovic, in a letter received since the above was written, has informed me that in an article published in 1905 (which I have not yet had an opportunity of consulting) he expressed the view that, besides other factors (trauma during operation, arterio-sclerosis, &c.), hypersecretion is necessary for the production of ulcer after gastrojejunostomy.

that at least the presumption is, not that the gastric contents were normal, but that they were hyperacid. I do not, however, press this point; I wish merely to indicate that this case cannot be used in argument to support the contention that jejunal ulcer occurs with normal gastric contents.

Let us now assume for a moment that jejunal ulcer may in exceptional cases occur without the presence of free hydrochloric acid in the jejunum. An explanation of such cases is not difficult. If we admit, as I think we must admit, that hyperacidity, or rather excess of free hydrochloric acid, is present so frequently in cases of jejunal ulcer as to justify the assumption that it is a cause of such ulceration, and if we admit that the hydrochloric acid acts by poisoning or killing the cells of the mucous membrane so that they are digested by the intestinal juices, we have to go only a step further and admit that other substances or toxins may, like hydrochloric acid, act as poisons and injure the living cells of the mucous membrane. Possibly of such an origin are the duodenal ulcers which sometimes accompany cutaneous burns or scalds, and more rarely complicate an attack of appendicitis. That toxic substances may be produced in the stomach we have presumptive evidence in the tetanic attacks which are sometimes associated with pyloric stenosis.

Let me briefly recapitulate the views set forth above: Jejunal ulcer following gastrojejunostomy is the result of a toxic agent or poison which so injures or kills the cells of the jejunal mucous membrane that they are readily digested by the intestinal juice. The toxic agent usually present is free hydrochloric acid, but possibly other toxic substances may be present, and either may increase the effect of the other. Thus, a small percentage of free hydrochloric acid in the jejunum, which by itself would not cause ulceration, may in the presence of some other toxic agent produce ulceration. The circumstances under which free hydrochloric acid may be present in the jejunum are:—

- (1) Hyperacidity of the gastric contents, so that the bile and pancreatic juice are unable to neutralize completely all the acid entering the jejunum.

- (2) Normal percentage of hydrochloric acid in the gastric juice but excessive secretion, so that the amount of hydrochloric acid discharged into the jejunum is greater than can be neutralized.

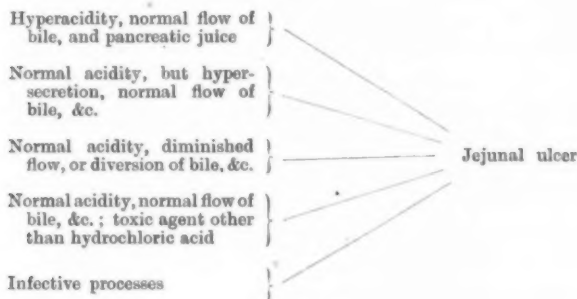
- (3) Diversion of the course of the bile and pancreatic juice, so that part of the jejunum is exposed to the action of the gastric contents unmixed with bile and pancreatic juice, as after operations of the "Y type," and gastrojejunostomy with entero-anastomosis.

(4) Normal acidity and amount of gastric secretion, but incomplete neutralization in the jejunum owing to temporary diminution of the flow of bile and of the secretion of pancreatic juice.

In a few cases jejunal ulcers are probably of infective origin. Such cases present the following features: (a) The ulcers occur within a very short period after gastrojejunostomy. (b) The ulcers are usually multiple.

Schematically, the ætiology of jejunal ulcer may be represented thus:—

SCHEME OF CAUSES OF JEJUNAL ULCER.



Since the foregoing part of this paper was written Dr. Charles Bolton [2] has published a most instructive and suggestive article, containing a record of observations which appear to me to support the views set forth above. Dr. Bolton found that feeding guinea-pigs on solutions of hydrochloric acid up to 0·7 per cent. produced no effect whatever, but solutions above this strength produced gastric lesions identical in character with those produced by a gastrototoxic serum. He found that the effect of hyperacidity combined with the injection of gastrototoxic serum was a marked increase of the stomach lesions. "It appears then," writes Dr. Bolton, "that any strength of hydrochloric acid above the normal can act as a protoplasmic poison for the gastric cells, and will add its quota to other devitalizing influence and assist in bringing about self-digestion. I think this assistance to produce lesions of poisons which are in themselves innocuous is a very important point. . . . That hyperacidity of the gastric juice increases the lesions produced by gastrot toxin, by the hydrochloric acid acting as a protoplasmic poison, and not by increasing the peptic activity of the gastric juice, is rendered probable by the fact that only within small

limits will an increase of hydrochloric acid assist in the digestion of foodstuffs." I think we may fairly argue that these conclusions as to the action of hydrochloric acid on the gastric mucous membrane may be applied with even greater force to the action of hydrochloric acid on the jejunal mucous membrane, which under normal conditions is unused to the presence of acid gastric contents.

So far I have discussed the ætiology of jejunal ulcer proper. Much of what has been said applies also to gastrojejunal ulcer. Hyperacidity is an important factor in both, but there is this difference: jejunal ulcers are a result of altered physiological conditions produced by operation; gastrojejunal ulcers are probably a direct consequence of operation. When a wound becomes ulcerated, the presumption is that the ulceration is a consequence of the wound. Beyond the general rule that jejunal ulcers occur most commonly in that part of the jejunum in which the acidity of the intestinal contents is greatest, we do not know what determines the site and area of ulceration. The site of a gastrojejunal ulcer is determined by the wound made in effecting the anastomosis, and the ulceration persists and spreads if there be hyperacidity of the gastric juice. In six of the fourteen cases of gastrojejunal ulcer the condition of the gastric contents is noted, and in five of these cases there was marked hyperacidity, and in the remaining case there was motor insufficiency and hypersecretion.

In connexion with the origin of gastrojejunal ulcer, the question arises, Does primary union take place between the mucous membranes of the stomach and jejunum when they are maintained in apposition by the use of an inner suture? It is obvious that the necrosis produced by a Murphy's button must result in a ring of ulceration which heals by cicatrization.¹ Non-apposition of the gastric and jejunal mucous membranes, injury to the mucous membrane during the operation, or infection of the suture used for approximation must also result in healing by granulation. A. H. Gould and Harrington [7], as the result of experiments on animals, conclude that an inner layer of stitches has little influence upon the healing of the wound. They found that whether the mucous membranes were apposed by suture or not, the mucous membrane round the margin of the anastomosis sloughed, and the separation of the slough left an ulcer which became covered over

¹ The process of repair following the use of Murphy's button has been investigated by Barbat, and F. T. Murphy has described the process of healing after anastomosis by means of McGraw's elastic ligature. A. H. Gould, in the work to which I refer later, gives an admirable summary of these researches.

with mucous membrane in about three weeks. If this be true, then it follows that every gastrojejunostomy is followed by a gastrojejunal ulcer. If hyperacidity or hypersecretion exist, then the ulcer, instead of healing, may persist or spread, and eventually cause symptoms such as have been described in an earlier part of this paper.

I must, however, state my belief that in human beings primary union of the gastric and jejunal mucous membrane may occur. To attain such a result rigid asepsis is imperative, an impossibility in operations on animals. In human beings, on the other hand, by adequate preparatory treatment and by careful dieting, it is possible to render sterile the upper part at least of the gastro-intestinal tract, and so to operate under conditions more favourable for primary union than can be obtained in experimental work on animals. I have had an opportunity of examining a considerable number of anastomoses from patients who have died at various intervals after gastrojejunostomy. In all I found direct continuity between the gastric and jejunal mucous membrane, and, further, the thread used for suturing was encapsuled and had not been discharged into the intestine. In several instances microscopical preparations from recent anastomoses showed continuity of mucous membrane, and no evidence of the sloughing which Gould states is the invariable rule. Whether, however, primary union be possible or not, we should employ the means most calculated to obtain it, and of this at least I am convinced—that the use of an inner suture ensures an opening which subsequently contracts less than an anastomosis effected by a button, or by the use of serous sutures only.

The regurgitation of bile and pancreatic juice which takes place into the stomach after simple gastrojejunostomy must be favourable to the union of the apposed surfaces by diminishing the acidity of the gastric contents as they pass through the opening, and Nature in her wisdom has so ordered it that this regurgitation is at its maximum just when it is most needed—in the early days after operation. This protective regurgitation is, so far as I have observed, absent in operations of the "Y type" and when an entero-anastomosis is performed.

Do the clinical histories of the recorded cases support the view that gastrojejunal ulcers are a direct consequence of operation? As I have pointed out already, we have no means of determining the date of the beginning of gastric or intestinal ulceration, and it seems certain that such ulcers may exist for a considerable time without causing symptoms. In view of this knowledge it is significant that in nine of the fourteen cases of gastrojejunal ulcer symptoms appeared within a

year. In two of the cases (Group I, No. 15, and Group II, No. 7) the interval between gastrojejunostomy and symptoms of ulcer was seven and eight years respectively. Any explanation of these late cases must be purely speculative. If it be true that pain is absent until ulceration invades the peritoneum, it is possible that an ulcer commencing as the result of gastrojejunostomy may be so chronic in its course that symptoms do not appear for many years. Another explanation may be suggested. Gastrojejunostomy may be followed by ulceration round the anastomotic opening; this may gradually heal, and in so doing cause some contraction of the opening. For a time the stomach is able to overcome the partial obstruction at the opening, but later the gastric muscle becomes, as it were, worn out, the stomach is no longer able to empty itself completely, and consequently stagnation and hyperacidity supervene. It is noteworthy that in both these very late cases there was marked hyperacidity with dilatation of the stomach.

The Closure of Gastrojejunostomy Openings.

In two of the cases (Group II, Nos. 6 and 29) under review there was complete, and in two other cases (Group I, No. 15, and Group II, No. 20) almost complete, obliteration of the gastrojejunostomy opening. Schostak [20] makes the suggestion that in most of the reported cases of closure of gastrojejunostomy openings the closure has been due to peptic ulceration. I am not aware that anyone has made this suggestion before, and yet it is obvious that closure of the opening must be the result of cicatrization following ulceration. Under what circumstances does this ulceration ensue? The reply to this query is correlated with the answer to another, Why is closure of gastrojejunostomy openings so infrequent nowadays? There must be some change in technique to account for this. I believe the explanation is that now the necessity for a large opening is becoming recognized, and that now almost all surgeons suture the mucous membrane of the stomach to that of the jejunum, whereas formerly they were content with a single layer of serous sutures, or used appliances such as Senn's plates or Murphy's button, which by compression cause a localized necrosis, resulting in a granulating surface which subsequently becomes contracted in the process of healing. In my Hunterian Lectures [15] I gave brief details of nineteen cases in which obliteration of a gastrojejunostomy opening had occurred, and it is significant that in seventeen of them either Senn's plates, Murphy's button, or a single layer of serous sutures had

been employed, all of which methods must lead to the formation of a considerable area of granulating surface. Now, one of two events may happen: either the granulation tissue may become cicatrized in the course of a few weeks, in which the diminution of the size of the opening will not be marked, or, on the other hand, the area of ulceration, instead of healing, may extend, so that if cicatrization subsequently occurs there will be marked contraction, or even obliteration, of the anastomotic opening. And just as hyperacidity is the condition which prevents healing of a gastric ulcer, so there can be little doubt that hyperacidity plays the chief role in the prevention of the healing of the granulating area round a recent gastrojejunostomy opening.

A few years ago we heard a good deal about the closure of gastrojejunostomy openings in cases of patent pylorus. Indeed, some surgeons went so far as to advocate that when gastrojejunostomy was performed in such cases, the pylorus should be deliberately excluded by suturing—a measure based on erroneous pathology, and little calculated to prevent closure of the anastomotic opening. The train of reasoning was that when the pylorus is patent, the food passes out through the pylorus in preference to the gastrojejunostomy opening, and so the artificial opening, as it is not used, gradually contracts or becomes completely closed. Now I venture to think that closure of the gastrojejunostomy opening occurs, not because the pylorus is patent, but because the cases in which the pylorus is patent are usually cases of gastric ulcer with marked hyperacidity. The hyperacidity is the causal factor, not the patent pylorus. It is in such cases that gentle handling of the mucous membrane, careful application of sutures, rigid asepsis, and strict dieting after operation are especially important, in order to prevent the occurrence of gastrojejunal ulcer with its attendant risk of subsequent contraction of the opening. I recognize so fully the great impetus which the introduction of Murphy's button gave to intestinal surgery that I hesitate to say any word which may seem to underrate the merit of this ingenious appliance; nevertheless I venture to think that its use is contra-indicated in cases of patent pylorus because of the hyperacidity usually existent.

The Influence of the Type of Gastrojejunostomy on the Causation of Jejunal Ulcer.

If jejunal ulcer be the result of the toxic action of hydrochloric acid on the jejunal mucous membrane, it follows that operations of the "Y type" are wrong in principle. After this type of operation the bile

and pancreatic juice enter the jejunum some inches below the anastomosis, so that this and several inches of jejunal mucous membrane are exposed to the action of unneutralized gastric contents. Monprofit, one of the few advocates of this operation at the present time, has obtained good results from it; nevertheless, I think that it is a method which should be employed, if at all, only in those cases in which free hydrochloric acid is absent from the gastric contents. Primary entero-anastomosis, an unnecessary and now seldom performed complication of gastrojejunostomy, is open to the same objection as gastrojejunostomy "En—Y." Inasmuch as the relative frequency with which these methods have been employed is unknown, we cannot prove that they are followed by jejunal or gastrojejunal ulcer more frequently than simple anterior or posterior gastrojejunostomy. But if, as is almost certainly the case, operations of the "Y type" (including entero-anastomosis) have been far less commonly performed than the simple anterior or posterior method, it is perhaps not without significance that in 24 per cent. of the recorded cases, jejunal or gastrojejunal ulcer has followed gastrojejunostomy "En—Y" or gastrojejunostomy with entero-anastomosis.

A perusal of the recorded cases shows that a large proportion have followed the anterior operation, and it has been argued that this method predisposes to jejunal ulceration. At first sight this appears to be the case, but I do not think that a closer examination of the records justifies this assumption. In making a comparison we should, for reasons given above, omit those cases in which an entero-anastomosis was performed. If we do this we find that in twenty-six instances jejunal or gastrojejunal ulcer followed a simple anterior operation, and in twelve instances a simple posterior operation. There is no doubt that at the present day the posterior operation is far more frequently performed than the anterior, but this was not the case some years back. I think we may fairly state that at any rate until the year 1901 the anterior operation was the more commonly performed. Now, if we divide the recorded cases into two groups—those which occurred after a gastrojejunostomy performed before the year 1901, and those which followed a gastrojejunostomy performed in 1901 or after—we find a marked difference in the proportion of cases following the anterior operation in the two periods, as is shown in the following table:—

TABLE XIV.

Gastrojejunostomy		Ulcer after	
		anterior operation	posterior operation
before 1901	...	16 cases	...
" in 1901 or after	...	6 "	6 "

It is possible that the upper part of the jejunum is more resistant than the lower to the toxic action of hydrochloric acid, and it is interesting to note that so far no instance of jejunal ulcer after a no-loop gastrojejunostomy has yet been recorded.¹ Perhaps this immunity of the no-loop operation may be due, not to any difference in the physiology of this method, but to improvements in the technique and treatment of gastric operations in general. I have already alluded to the infrequency of jejunal ulcer in America, in the days of both loop and no-loop operations, and my impression is that this is a consequence of the high pitch of perfection to which our transatlantic brethren have brought their operative and aseptic technique. Be this as it may, it is hardly likely that the resisting power of the jejunum at the site of a posterior gastrojejunostomy with a loop differs so materially from the resisting power at the site of an anterior operation as to account for the difference in the incidence of jejunal ulcer. The observation that since 1901 the number of cases of jejunal ulcer which have followed the anterior and posterior operations is the same, suggests that the greater frequency of jejunal ulcer following the anterior operation is simply because in the early days the anterior was the more commonly performed.

The Causes of Hyperacidity after Gastrojejunostomy.

I have already pointed out that after gastrojejunostomy there is a marked fall in the total acidity of the gastric contents, the result of the altered physiological conditions affected by the operation. This diminution varies from 25 per cent. to 40 per cent., and, as a rule, is sufficient to convert hyperacidity before operation into hypo-acidity after operation. What, then, are the causes of hyperacidity after operation? We must draw a clear distinction between two classes of cases:—

- (1) Cases in which hyperacidity is present before, and *persists* after operation.
- (2) Cases in which after a period of normal or subnormal acidity following operation the gastric contents subsequently become hyperacid.

(1) HYPERACIDITY PERSISTS AFTER GASTROJEJUNOSTOMY FOR ONE OF TWO REASONS:—

(a) *Extreme Hyperacidity before Operation.*—I have estimated the acidity of the gastric contents before and after operation in fifty-six

¹ Moynihan mentions a case in which adhesions round the jejunum were regarded as indicating the existence of a jejunal ulcer.—*Surg., Gynecol., and Obstet.*, Chicago, 1907, iv. pp. 683-4.

patients on whom I have performed gastrojejunostomy, and I find that the average diminution of the acidity after operation is 33 per cent. If before operation the total acidity is over 100, then the diminution following operation is sometimes insufficient to reduce the acidity of the gastric contents to normal, unless the patient is kept on a milk diet for some months.

(b) *A too Small, Inefficient, or Defective Anastomotic Opening.*—To secure the best physiological results two things are in my experience of importance—first, to make a large opening; and, secondly, to make the opening towards the pyloric end of the stomach.

(2) HYPERACIDITY OF THE GASTRIC CONTENTS SUCCEEDING A PERIOD OF NORMAL OR SUB-NORMAL ACIDITY IS PROBABLY DUE TO ONE OF TWO CAUSES—TOO SMALL AN OPENING, OR INDISCRETIONS IN DIET.

In some cases both these factors may be present. When gastrojejunostomy is performed on a dilated stomach, the operation is usually followed by a considerable diminution of the size of this organ, and so it may happen that an opening originally of good size may become too small owing to the contraction of the stomach around it. Hayem [8] states that the presence of an acid liquid in a fasting stomach is an indication that the opening does not allow of complete evacuation of the stomach. A small or inefficient opening leads to gastric stasis, gastritis, and hyperacidity. I quite admit that a slight degree of gastric stasis may have no ill result; but it is reasonable to argue that in such a case a slight attack of gastritis due to some error in diet, insufficient to cause trouble if the anastomotic opening were larger, may lead to such a degree of gastric stasis that hyperacidity ensues. Doubtless in some cases long-continued indiscretions in diet may lead to gastritis, however large the opening may be, and in this connexion it is interesting to note that one of the fatal cases recorded (Group I, No. 10) is that of a man known to have been a heavy drinker, and the ulcer perforated while he was drunk.

I have observed hyperacidity after gastrojejunostomy in three cases. In one the hyperacidity before operation was so great that notwithstanding a diminution of nearly 40 per cent., hyperacidity still persisted after operation, but gradually fell below normal as the result of careful dieting. In the second case, in which gastrojejunostomy had been performed some months previously by another surgeon, subsequent operation

showed that the opening between the stomach and jejunum was so extremely small that I had to make a new opening. While the third case is the one which I have reported in full at the beginning of this paper. In this case I am of opinion that indiscretions in diet played the chief part in causing hyperacidity; but at the same time I think the opening was of insufficient size, and it will be noted that in this case, one of my earlier gastrojejunostomies, a single layer of Halsted's sutures was employed, but no inner sutures.

Let us now briefly review the cases to see in what proportion the conditions I have mentioned as favouring jejunal and gastrojejunal ulcer were present. The patients in whom perforation peritonitis occurred were not in a condition for any elaborate clinical investigation, so our inquiry is limited to the cases of Group II. Details are available in twenty of the cases. Hyperacidity was present in eight cases, an entero-anastomosis had been performed in eight cases, and in five of these hyperacidity was present in addition, and in one there was hypersecretion. Gastrojejunostomy "En-Y" was performed in one case; in one case there was evidence of obstruction at the anastomotic opening; and in one case in a cavity surrounding the anastomosis, a piece of silk was found which presumably acted as a septic focus. In one case it is stated that the gastric acidity was normal. We see thus that in nineteen of the twenty cases in which details are given, one of the conditions mentioned as favouring secondary ulcer was present, and in six of these cases more than one of the conditions referred to was present. These observations put in tabular form are as follows:—

TABLE XV.

Hyperacidity	8 cases
Entero-anastomosis	2 "
"	+	hyperacidity	5 "
"	+	hypersecretion	1 case
Gastrojejunostomy "En-Y"	1 "
Evidence of obstruction at anastomotic opening	1 "
Septic focus	1 "
Normal gastric acidity	1 "
					—
					20

The Ultimate Results of Operation for Jejunal and Gastrojejunal Ulcer.

The remote results of the surgical treatment of jejunal and gastrojejunal ulcers do not present a pleasing picture. I have been able to ascertain the later history of twenty-five of the thirty-one patients who

recovered from operation. Of these twenty-five, no fewer than fifteen (60 per cent.) relapsed, two of whom died later after two or more operations for recurrence of symptoms (Group II, Nos. 2 and 23). Seven of the patients who suffered from relapse are known to have been free from further trouble for periods varying from nine months to nine years. Ten only of the patients can be classified as "cured," and even this is perhaps an optimistic conclusion, as in some of these ten patients the interval which has elapsed since operation up to the time of the latest report is under one year. Putting the most favourable construction on these figures, we see that 60 per cent. of the patients have suffered relapse, 40 per cent. have been apparently "cured" by one operation, and 28 per cent. after two or more operations. These results are shown in the following table:—

TABLE XVI.—LATE RESULT OF SURGICAL TREATMENT OF JEJUNAL AND GASTRO-JEJUNAL ULCER.

"Cured"	10
Relapsed:					
Died after two or more operations	...				2
Well	"	"	"	...	7
Not cured	"	"	"	...	3
Later result unknown	3
				—	15
					25

What can we learn from a study of the different methods of treatment employed in these cases? First, I think they teach us that the simpler the surgical procedure employed, the better the result. Resection of intestine, combined with gastrojejunostomy "En—Y," has not been attended with satisfactory results, and the same criticism applies to the cases in which an entero-anastomosis has formed part of the treatment. In just over half of the cases classified as "cured" simple suture only of the ulcer was the method adopted, followed doubtless by a course of careful dieting. Put in tabular form, the facts are as follows:—

TABLE XVII.—RESULTS OF VARIOUS METHODS OF SURGICAL TREATMENT.

	Cured	Relapsed
Suture only (11 cases) ...	6	5
New gastrojejunostomy and suture (4 cases) ...	3	1
Resection and gastrojejunostomy "En—Y," or suture and entero-anastomosis (3 cases) ...	1	7

Secondly, we learn that those cases, so far as the data available enable us to judge, do best in which, at the time of operation for jejunal or gastrojejunal ulcer, hyperacidity does not exist. Hyperacidity or

hypersecretion is not favourable to the healing of the extensive wounds left by resections of intestine and gastrojejunostomy "En—Y." In my own case, I attribute the comparatively successful result obtained to the circumstance that before operation the patient was kept on a milk diet until the hyperacidity had markedly diminished. Of the five cases in which relapse occurred after simple suture of the ulcer, ultimate success was obtained in one by simple suture of the second ulcer, and in two by the performance of a new gastrojejunostomy. The foregoing facts seem to indicate that part of the surgical treatment of jejunal and gastrojejunal ulcers should consist either in the enlargement of the original gastrojejunostomy, or in the making of a new one. Whether this should be done at the same time as the suture of the ulcer, I will discuss later.

The Treatment of Jejunal and Gastrojejunal Ulcers.

The treatment of the cases in which perforation into the general peritoneal cavity occurs is obvious. Immediate laparotomy and suture of the perforation offer the only prospect of saving the life of the patient. If the patient recover, the condition of the gastric contents should be investigated, and if hyperacidity, hypersecretion, or gastric stasis exist, the gastrojejunostomy opening should be enlarged, or a new gastrojejunostomy performed.

In the cases of Group II in which protective adhesions are present, the indication for surgical treatment is not so clear. The results of operation are not such as to encourage us to resort to surgery until after a trial of medical treatment. Thus, in one case (Group II, No. 1) four operations, in four cases (Group II, Nos. 2, 9, 12, and 22) three operations, and in seven cases (Group II, Nos. 3, 5, 6, 8, 13, 19, and 23) two operations were necessary; and of these twelve patients, two subsequently died and three were no better after operation than before.

In connexion with medical treatment the question arises, What evidence have we of the spontaneous healing of jejunal and gastrojejunal ulcers? We have already seen that obliteration of the gastrojejunostomy opening occurred in two cases (Group II, Nos. 6 and 29), and that this obliteration must have been the result of cicatrization following ulceration. It is true that in those cases surgical intervention was subsequently necessary, nevertheless they show that ulceration at the site of the anastomosis may, under some circumstances, heal spontaneously. In some of the cases in which the presence of ulcer was

demonstrated at operation, we have indirect evidence to the same effect. In one case (Group II, No. 11), six months after operation for jejunal ulcer the patient suffered relapse and a tender, hard swelling formed in the region of the cicatrix. Under medical treatment the patient became quite well, although whether this improvement was permanent or not I have been unable to ascertain. In another case (Group II, No. 19), after two operations for jejunal ulcer, relapse occurred, but after a time the patient improved, and two years later was in better health than for many years. In my own case relapse with hyperacidity occurred, and the presence of blood in the stomach rendered the existence of ulceration highly probable, but under dietetic and medical treatment the symptoms gradually disappeared.

In this connexion a perusal of the cases classified as "doubtful" is of interest, although their value as evidence is somewhat dubious. Thus, in Fraenkel's case (No. 1A) an intestinal fistula formed, probably the result of a jejunal ulcer. Gradual closure of the fistula ensued. In Tiegel's case (No. 2A), two years after gastrojejunostomy with entero-anastomosis, the presence of severe pain and a tender resistance rendered the existence of a jejunal ulcer highly probable. Under treatment the patient improved, but it is important to note that after a few months of good health the attacks of pain recurred. In Hahn's cases (Nos. 3A and 4A) the details given are so scanty as to be of little service. In Cackovic's case (No. 5A) the patient suffered from spasmodic pain and a painful swelling of the abdominal wall. After some months' treatment the patient recovered, and Dr. Cackovic has kindly informed me that seventeen months later she was quite well. In Lyle's case (No. 6A) symptoms of ulcer recurred, which were relieved by medical treatment. In Schostak's case (No. 7A) a tender infiltration in the umbilical region, accompanied by pain, gradually disappeared under treatment. In my doubtful case (No. 8A), after separations round a presumed jejunal ulcer, the patient remained comparatively well for over two years.

Brodnitz's case (Group II, No. 13) is quoted by Schostak as an instance of cure under medical treatment, but the further details with which Dr. Brodnitz has kindly furnished me show that the patient subsequently required further operative treatment. I have pointed out already that examination of the specimen from my own case showed that the jejunal ulcer had healed before I operated, and in another case (Group II, No. 31) the fistula between the jejunum and colon was lined with mucous membrane which showed no trace of ulceration. Although, in both these cases, the ulcer healed, they can hardly be regarded as

instances of cure without operation, as in both surgical interference was imperative.

The evidence, therefore, as to the possibility of permanent relief by medical treatment is very inconclusive. In two only of the cases so treated is the later history known, and in one of these (Group II, No. 13) relapse subsequently ensued. All we can say for certain is, that occasionally jejunal and gastrojejunal ulcers heal, but the process of healing is apt to result in a condition requiring surgical intervention.

MEDICAL TREATMENT.

Notwithstanding the conclusions formed from a study of the cases, I think that before an operation is performed, the patient should undergo a course of careful dietetic and medical treatment for two reasons: first, there is just a possibility—remote, it is true—that in this way a cure may be brought about; and, secondly, if subsequently operation be necessary, success will be more likely to follow if, as the result of medical treatment, the acidity of the gastric contents is diminished.

The patient should be kept at absolute rest in bed, and on a diet of milk and eggs, as recommended by Lenhartz for the treatment of gastric ulcer. Bismuth, combined with small doses of hydrocyanic acid, usually relieves the pain, and the researches of Pawlow [16] appear to me to show conclusively the value of the administration of alkalis in diminishing the acidity of the gastric contents. The effect of diet in reducing the acidity of the gastric contents is well illustrated in my own case, as well as in the cases (Group II, Nos. 11 and 24) reported by Mikulicz and Einar Key. If, under medical treatment, the pain disappears and the gastric acidity becomes diminished, we may persevere in the hope of ultimate cure, and the treatment should be prolonged for six months at least. The two indications for surgical treatment appear to me to be: first, persistence of pain and hyperacidity in spite of treatment; and, secondly, evidence of hypersecretion, or gastric stasis. The presence of these conditions indicates that the anastomotic opening is at fault, and under these circumstances there is little likelihood of complete and permanent cure without surgical intervention. If, in the early morning, after ten hours' abstention from food, an acid fluid can be obtained from the stomach, this is sufficient indication of the necessity for surgical treatment.

SURGICAL TREATMENT.

This must necessarily depend on the condition found at operation. Adhesions must be separated, and the perforation, if there is one, sutured. A study of the recorded cases plainly indicates that the less extensive the operation performed, the better is the result, especially if marked hyperacidity exist. The anastomotic opening should be carefully examined, and if it is small it should be enlarged, or a new gastro-jejunostomy performed. The former is, in my judgment, the preferable course, as being not only simpler, but does not necessitate a "Y type" operation, which, for reasons already given, is not to be recommended. If, on account of severe pain or rapid loss of strength, operation has to be undertaken before an attempt has been made to reduce any hyperacidity which is present, I would suggest that the enlargement of the gastrojejunostomy should be deferred; but if, on the other hand, a strict course of medical treatment has been ineffectual in reducing acidity, then no object is gained by deferring the completion of whatever operative procedure is requisite.

In the cases, unfortunately too numerous, in which, after surgical treatment, the symptoms recur, I suggest that a gastrojejunostomy "En-Y" should be performed, but with implantation of the proximal limb of the jejunum into the stomach, so that the bile and pancreatic juice are diverted directly into the stomach.

PREVENTIVE TREATMENT.

Our great aim, however, should be the prevention of jejunal or gastro-jejunal ulcer, and I will very briefly indicate, under two headings, what appear to me the points of importance in this connexion:—

- (1) The technique of gastrojejunostomy.
- (2) The after-treatment.

(1) *The Technique of Gastrojejunostomy.*

First, I would emphasize the necessity for a large opening. There is invariably some contraction of the opening, and in cases of dilated stomach subsequent diminution in the size of this viscus still further narrows the communication between the jejunum and stomach, and it is, perhaps, not without significance that in thirty-three of the recorded cases the gastrojejunostomy was performed by pyloric stenosis.

Secondly, accurate apposition of the mucous membrane of the stomach and jejunum, so as to secure, if not primary union, at any rate union with the formation of a minimum of scar tissue.

Thirdly, careful application of the inner suture so as to control bleeding, but, at the same time, to avoid such a degree of tightness as will cause localized necrosis of the tissues.

Fourthly, the use of simple suturing in preference to mechanical appliances, especially when hyperacidity is present.

Fifthly, the avoidance of entero-anastomosis and of "Y-type" operations.

It has been suggested that catgut should be used for the inner suture in preference to silk or linen, because the latter is more apt to serve as a septic focus. This appears to have been the case in one case (Group II, No. 23). I have already alluded to the importance of reducing acidity and rendering the gastric contents sterile, as aids in securing primary union, and I may add that personally I prefer the greater security of a linen thread to the use of catgut.

Gastrojejunostomia fundosa, as recommended by Goepel, does not, in my experience, give such good results as when the anastomotic opening is placed near the pyloric end of the stomach.

(2) *The After-Treatment of Gastrojejunostomy.*

The necessity for prolonged after-treatment in cases of gastrojejunostomy has, perhaps, not received the attention which it deserves. My rule is to advise all patients whose gastric contents have been hyperacid before gastrojejunostomy, to avoid meat in any form for six months at least, and until such time as examination shows that the gastric acidity is sub-normal. The immediate relief which is experienced by patients on whom gastrojejunostomy has been performed tempts them to indulge in food unsuited to the condition of the gastric mucosa. In most cases in which gastrojejunostomy is necessary, the mucous membrane is chronically inflamed, and many months must elapse before it is restored to a healthy condition.

Some surgeons, in their dread of jejunal ulcer, have maintained that gastrojejunostomy is contra-indicated in gastric ulcer with hyperacidity, except when the ulcer is near the pylorus and is causing symptoms of obstruction. Others have even suggested that unless there be gastric stasis, gastrojejunostomy is useless in the treatment of gastric ulcer. I

believe this teaching to be retrogressive. For some years I have been advocating the view that gastrojejunostomy is not a drainage operation. The success which follows this operation in cases of gastric ulcer is due, not to drainage, but to the physiological effects of the operation in diminishing the acidity of the gastric contents, and this diminution follows gastrojejunostomy irrespective of the situation of the ulcer.

Summary of Conclusions.

(1) The risk of jejunal ulcer following gastrojejunostomy is probably under 2 per cent.

(2) At the present time this complication apparently occurs less frequently than formerly.

(3) Clinically, there are two groups of cases: (1) those in which perforation into the general peritoneal cavity ensues; (2) those in which general peritonitis is prevented by the formation of adhesions.

(4) Pathologically the cases may be classified as follows: (1) ulcers of the jejunum; (2) gastrojejunal ulcers, or ulcers at the site of the anastomosis.

(5) Jejunal ulcers in some instances are of infective origin; in these cases ulceration commences within a very short interval after gastrojejunostomy, and usually the ulcers are multiple.

(6) In a large proportion of cases the ulcer is single, and is probably the result of the toxic action of hydrochloric acid, which injures the cells of the mucous membrane so that they are digested by the intestinal juice. Possibly other agents than hydrochloric acid may play a part in injuring the mucous membrane.

(7) Gastrojejunal ulcers are a direct consequence of the wound made in effecting the anastomosis, and their persistence is probably the result of hyperacidity of the gastric juice.

(8) Closure of a gastrojejunostomy opening is the consequence of cicatrization of a gastrojejunal ulcer. It is more likely to occur when the pylorus is patent, not because of the patency of the pylorus, but because in such cases hyperacidity is usually markedly present.

(9) Any procedure or disease which diminishes the amount of bile and pancreatic juice in the jejunum favours the occurrence of jejunal and gastrojejunal ulcer. For this reason operations of the "Y type" and entero-anastomosis are inadvisable, at any rate in cases in which free hydrochloric acid is present in the gastric contents, as after these

procedures the anastomosis and a portion of the jejunum are deprived of the protective influence of the alkaline bile and pancreatic juice.

(10) The reason that ulceration has followed the anterior operation more frequently than the posterior operation with a loop, is probably that in former times the anterior operation was more frequently performed.

(11) As no instance of ulcer after the posterior no-loop operation has yet been recorded, we must for the present assume that its occurrence after this type of operation is less likely. It is possible, however, that this immunity is partly the result of improvements in technique and in the after-treatment of gastric operations in general.

(12) In cases in which perforation into the general peritoneal cavity occurs, immediate laparotomy offers the only chance of saving the patient's life.

(13) Inasmuch as there is some evidence that jejunal and gastrojejunal ulcers may heal, an operation should not be performed in the chronic cases until after a thorough trial of medical treatment.

(14) Even when surgical intervention is necessary an attempt should first be made to diminish hyperacidity, if this be present.

(15) Our aim should be to prevent the occurrence of this complication of gastrojejunostomy. Preventive treatment consists in (1) careful and appropriate surgical technique and (2) prolonged after-treatment.

(16) Lastly, every case of recrudescence of pain of a constant character after gastrojejunostomy, especially when associated with hyperacidity or hypersecretion, should be regarded as a case of potential ulcer, and treated accordingly.

Table XVIII.—Abstract of Cases of Jejunal Ulcer.

The numbers in first column refer to the group and number of the cases as detailed in the Paper (pp. 249-67).
Group I: Cases in which general peritonitis occurred. Group II: Cases in which general peritonitis was prevented by adhesions.

No.	Recorder	Sex	Age	Method of gastrojejunostomy	Interval between gastrojejunostomy and appearance of symptoms	Interval between gastrojejunostomy and second operation	Operative treatment	Result
I-1	Braun	M.	25	Posterior	11 months	—	No operation	Death
I-2	Hahn	M.	?	Anterior	1 year	—	No operation	Death
I-3	Kürte	M.	30	Anterior	3 years	3 years	Diagnosed as appendicitis	Death
I-4	Steinthal	M.	44	Posterior (Murphy)	10 days	—	No operation	Death
I-5	Goepel	M.	?	Anterior	13 months	—	No operation	Death
I-6	Goepel	M.	?	Anterior	4 months	4 months	Suture of perforation	Recovery
I-7	Goepel	M.	34	Anterior	9 months	9 months	Suture of perforation	Recovery
I-8	Tiegel	F.	2 months	Anterior, with entero-anastomosis	2 months	—	No operation	Death
I-9	Edington	M.	39	Anterior	7 years	7 years	Suture of perforation	Death
I-10	Peterson	M.	48	Anterior	2 years	—	No operation	Death
I-11	Peterson	M.	51	Anterior	24 years	—	No operation	Death
I-12	Hamann	M.	48	Anterior, with entero-anastomosis	26 days	—	No operation	Death

No.	Recorder	Sex	Age	Method of gastrojejunostomy	Interval between gastrojejunostomy and appearance of symptoms	Interval between gastrojejunostomy and second operation	Operative treatment	Result
I-13	Battle	M.	30	Anterior (Murphy)	22 months	22 months	Suture of perforation	Recovery
I-14	Battle	F.	30	Anterior	1 year	1 year	Suture of perforation	Recovery; relapse; third operation; cure
I-17	Key	F.	25	Anterior (partial gastrectomy)	10 days	—	No operation	Death
I-18	Cackovic	M.	30	Posterior	2 days	—	No operation	Death
II-2	Tiegel	M.	25	Anterior, with entero-anastomosis	3 months	1½ years	Excision and jejunostomy	Recovery; death after fourth operation
II-3	Quénu	M.	29	Anterior (Murphy)	1½ years	4½ years	Excision and gastrojejunostomy "En-Y"	Recovery; relapse; jejunostomy; cure
II-4	Koehler	M.	—	"En-Y"	3 months	1 year	Excision of ulcer	Recovery
II-5	Heidenhain	M.	48	Anterior	? months	? months	Suture of perforation	Recovery; relapse; new gastrojejunostomy; cure
II-6	Kreuzer	M.	36	Anterior	1½ years	5½ years	Suture of perforation	Recovery; relapse; third operation; cure
II-9	Neumann	M.	24	Anterior, with entero-anastomosis	6 months	1 year	Excision of ulcer	Recovery
II-11	Tiegel	M.	33	Anterior, with entero-anastomosis	1½ years	4½ years	Suture of perforation	Recovery; relapse
II-13	Brodnitz	M.	56	Anterior	3½ years	8 years, 10 months	Resection and gastrojejunostomy "En-Y"	Recovery; relapse; third operation; cure

II-14	Robson	M.	44	Anterior	2 years	3 years, 4 months	Resection and gastrojejun- ostomy "En-Y"	Recovery
II-15	Jahr	M.	29	Anterior, with entero- anastomosis	8 months	1½ years	Excision of ulcer	Recovery
II-16	Hofmann	F.	22	Anterior, with entero- anastomosis	6 years	6 years	Excision of ulcer	Recovery
II-21	Cackovic	M.	30	Anterior	8 months	13 months	Suture of perforation	Recovery
II-22	Key	M.	39	Anterior	6 months	2½ years	Suture of perforation	Recovery; relapse
II-24	Key	M.	26	Anterior	4 years	5 years	Suture of perforation	Recovery
II-25	Key	F.	26	Posterior	6 weeks	7 weeks	Gastrojejunostomy "En-Y"	Death
II-26	Rotgans	M.	?	Anterior	?	?	Excision of ulcer	Recovery
II-27	Cackovic	M.	36	Anterior	3 days	16 days	Suture of perforation and entero-anastomosis	Recovery
II-28	Paterson	F.	47	Anterior	3 years	5 years	Resection of jejunum and gastrojejunostomy "En-Y"	Recovery; relapse; recovery
II-29	Kaufmann	M.	41	Anterior, with entero- anastomosis	3 months	3½ years	Closure of gastro-colic and jejuno-colic fistula	Death
II-31	Gosset	M.	40	Posterior	1½ years	22 months	Closure of jejuno-colic fistula	Recovery
II-32	Herczel	?	?	Posterior	?	?	Closure of jejuno-colic fistula	
II-33	Cackovic	M.	38	Posterior	3 years 2 months	—	Died 6 years after gastro- jejunostomy	Death; no operation

Table XIX.—Abstract of Cases of Gastrojejunal Ulcer (Ulcer at site of Anastomosis).

The numbers in first column refer to the numbers of the cases as detailed in the Paper. Group I: Cases in which general peritonitis occurred. Group II: Cases in which general peritonitis was prevented by adhesions.

No.	Recorder	Sex	Age	Method of gastrojejunostomy	Interval between gastrojejunostomy and appearance of symptoms	Interval between gastrojejunostomy and second operation	Operative treatment	Result
I-15	Graser	F.	?	Posterior (Murphy)	3 years	—	No operation	Death
I-16	Key	F.	45	Anterior "En-Y"	7 years	7 years	Suture of perforation	Recovery
I-19	Delaloye	M.	41	Anterior	3½ years	—	No operation	Death
II-1	Mikulicz	M.	32	Anterior	4 months	Over 4 months	Suture of perforation and entero-anastomosis	Recovery; relapse
II-7	Czerny	M.	59	Posterior	8 years	9 years	Excision of ulcer and gastrojejunostomy "En-Y"	Death
II-8	Czerny	M.	42	Posterior (Murphy)	6 months	1½ years	Partial gastrectomy	Death
II-10	Mikulicz	M.	53	Anterior with entero-anastomosis	8 months	4 years 4 months	Suture of perforation	Death
II-12	Schloffer	F.	31	Anterior with entero-anastomosis	3 months	1 year 3 months	New gastrojejunostomy	Recovery; relapse; later operation; cure
II-17	Hofmann	M.	45	Posterior	1 year	5 years	New gastrojejunostomy (Murphy)	Recovery
II-18	Schostak	M.	22	Anterior	1 year	2½ years	Suture of perforation, entero-anastomosis	Recovery
II-19	Schostak	F.	18	Anterior	1 year	1 year 10 months	Excision, gastrojejunostomy "En-Y"	Recovery; relapse; later operation; recovery
II-20	Connell	?	?	Anterior	2 years	3 years	New anterior gastrojejunostomy	Recovery
II-23	Key	F.	38	Anterior	3 months	2 years 8 months	Resection of jejunum, gastrojejunostomy "En-Y"	Recovery; relapse; operation; death
II-30	Czerny	M.	36	Posterior (Murphy)	1½ years	2 years	New gastrojejunostomy	Recovery

Table XX.—Abstract of "Doubtful" Cases of Jejunal Ulcer.

No.	Recorder	Sex	Age	Method of gastrojejunostomy	Interval between gastrojejunostomy and appearance of symptoms	Symptoms and signs	Result
1A	Fraenkel	?	?	Anterior with entero-anastomosis	11 months	Fecal abscess	Recovery
2A	Tiegel	M	41	Anterior with entero-anastomosis	2 years	Pain, resistance in abdomen	Recovery and relapse
3A	Hahn	?	?	?	?	Abscess of abdominal wall	Recovery
4A	Hahn	?	?	?	?	Abscess	Recovery
5A	Cackovic	F	40	Anterior	6 days	Pain, swelling of abdominal wall	Recovery
6A	Lyle	M	26	Posterior with entero-anastomosis	6 months	Symptoms of gastric ulcer	Relief
7A	Schostak	M	70	Anterior	1 year	Pain, tender induration	Recovery
8A	Hadra	M	?	Anterior	6 months	Pain, infiltration of abdominal wall, gastric fistula	Recovery
9A	Paterson	M	76	Anterior (Murphy)	5 years	Pain, swelling in left rectus, adhesions round jejunum	Gastrolysis; relief
10A	Nyrop	?	?	?	?	?	?
11A	Paterson	M	47	Posterior	4 years	Pain, resistance	?

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DISCUSSION.

Dr. C. BOLTON said that Mr. Paterson had referred to some work on which he (Dr. Bolton) had been engaged during the past six years, concerning the pathology of gastric ulcer. Two years ago he published a paper in the *Proceedings of the Royal Society*,¹ in which he showed that gastric ulcer might be brought about in one of two ways: either by some poison circulating in the blood, which initiated self-digestion by devitalizing the gastric cells, or, secondly, by some poison introduced into the cavity of the stomach acting in a similar way. And he found, as Mr. Paterson had mentioned, that hyperacidity of the gastric juice would act as a protoplasmic poison; that if a 0.7 per cent. solution of hydrochloric acid were introduced into the guinea-pig's stomach, ulceration was produced, but not with lower strengths. However, a strength as low as 0.3 per cent. was able to increase the ulceration when the stomach cells were devitalized by a poison circulating in the blood. In gastric ulcer one had first to consider the origin of the ulcer, and, secondly, why it did not heal. He had been reminded of the second point by some remarks of Mr. Paterson's. He was working on the matter now and had found that hyperacidity of the gastric contents did not prevent the healing of a gastric ulcer if the health of the body were normal, and if the stomach emptied itself in the normal time. He had also shown that diminished acidity of gastric contents, which it was possible to produce, might slightly delay the healing, though not very much. It was only a question of a few days. Therefore, by altering the acidity of the secretion alone it was not possible to produce a chronic ulcer. During the past few weeks he had shown that one could definitely delay the healing of an ulcer in the cat by pyloric constriction which led to motor insufficiency. The ordinary ulcer healed in three weeks, and by this means he had produced an ulcer seven weeks old. That he regarded as a very important matter from the point of view of both the physician and the surgeon. By constricting the pylorus, the healing of the ulcer was delayed. He thought that might to some extent account for the good results which

¹ *Proc. Roy. Soc. Lond.*, 1905-6, s. B., lxxvii, p. 426.

follow gastro-enterostomy. He had not yet discovered how long it was possible to delay the healing of the ulcers. With regard to the relation of hyperacidity of the gastric juice to ulcer, two views were held: first, that gastric ulcer, by reflex excitation, caused hyperacidity. He had been able to show that such was not the case for ulcers produced in the guinea-pig, at all events. In nineteen cases he had found that at the beginning, when the ulcer was in a sloughing condition, the active hydrochloric acid was diminished, and that this diminution was not due to neutralization of the acid by any alkaline discharge from the ulcer, because the inorganic chlorides of the stomach contents were not increased. Later, during the process of healing, it became normal, so that probably hyperacidity was not due to the mere presence of any ulcer. The other idea was that the hyperacidity was quite a separate condition and might play a part in the production of the ulcer, and that was the view he himself held. Hyperacidity was a common form of functional disturbance of the stomach and so was motor insufficiency, and he thought hyperacidity was one form of functional disease complicating gastric ulcer. With regard to treatment, he was not inclined to approve of Mr. Paterson's method of allowing the bile and pancreatic juice to flow into the stomach as a means of neutralizing the gastric juice: it would be better to give an alkali by the mouth. He thought jejunal ulcer might be directly due to hyperacidity, because the jejunum must have a less resisting power to hydrochloric acid than had the stomach; and then it was possible that a 0.3 or 0.25 per cent. strength might act as a protoplasmic poison and so devitalize the jejunal cells as to initiate self-digestion. Such a jejunal ulcer would not heal up so readily as a gastric ulcer.

Mr. W. G. SPENCER said that, with regard to hyperacidity, if the conditions were to be taken wholly as Mr. Paterson had stated them, it would tend to limit the application of the operation of gastrojejunostomy. The author seemed to lay rather too much stress on hyperacidity in some parts of his paper, because every operation of gastrojejunostomy entailed that acid juice passed into the jejunum. Most of the cases had followed operation done by the older methods, and the author had not mentioned many cases in which the newer operation had been used. In other words, the consequence of the various improvements in the technique of the operation had rather tended to banish that bugbear whenever the operation had been performed under the best circumstances. But in his (Mr. Spencer's) experience, even with the anterior form of gastrojejunostomy, he had not happened to meet with jejunal ulceration. He had thought it right to do an anterior gastrojejunostomy in a good number of cases, particularly in attempting to relieve carcinoma which had passed beyond the possibility of excision. At present, owing to the fact that the posterior operation got invaded by the cancer early, he thought that some patients, of late years, had not been operated upon. Especially in cancer, in which one presumed that before the patient's death something like half the stomach would be involved, if one did an operation on the cardiac side one might afford some months of considerable relief. Mr. Paterson would

say that it followed from his line of reasoning that, as in cancer there was diminution of acidity of gastric juice, therefore there was no likelihood of jejunal ulcer following. But he was arguing that as the anterior operation was most convenient the patient should be offered that operation, and that the danger of jejunal ulcer occurring after the operation was not at all a likely one. He knew it was said that jejunal ulcer particularly followed from the anterior operation. He, Mr. Spencer, had had over 40 cases in which the anterior operation was done, principally for cancer, in which the patients had been relieved on an average for some months; and in none of them had anything like a jejunal ulcer occurred. With regard to the primary union of mucous membrane, he had examined some of the cases which had died soon after the operation. The openings were something more than the inch in the long axis, the mucous membrane was adherent and he saw no sign of ulcer. They had been done with a buttonhole suture, or with Connell's stitch, which brought the mucosa very closely together. Therefore, he did not think there was now danger of the gastrojejunal type of ulceration such as occurred at the site of ulceration. Whatever might be said about some cases of failure of gastrojejunostomy, at any rate for cases of definite pyloric obstruction the posterior operation might be done, and for advanced cases of cancer the anterior might be done. In the one case the patient might be permanently relieved, and in the second case the patient might be relieved for months. And if those operations were done carefully, he thought the operation a good one. He admitted that gastrojejunostomy was thought to have been overdone, and was consequently somewhat under a cloud. But the sources of this view were broadly either mistakes of technique or from operations being done in cases of what physicians called nervous dyspepsia.

Mr. JONATHAN HUTCHINSON said the paper was so full and complex that it was difficult to take in all the main points. But he wished to ask about three matters. First, what was meant by "suture of the intestine" in Cases 10 and 17? Did it mean that an ulcer was inferred from the thinness of the intestinal wall and that that wall was then infolded by suture? Or was the ulcer excised? It might be either a very slight operation or a very serious one. With regard to the cases of jejunostomy which the author mentioned, apparently two or three cases of dyspeptic jejunal ulcer had been cured by jejunostomy, but no information was given as to what was meant. Was the patient actually fed through the jejunal opening, or was a fistula established on the surface? In either case the proceeding did not seem to be one of the simplest, and Mr. Paterson had advised that any operative treatment in these cases should be of a simple character. Then Mr. Paterson's proposal in those cases was to divert the bile and carry that and the pancreatic juice back into the stomach as a cure for the condition. That was an interesting proposal, but he gathered that it had not been actually carried out. It seemed open to the drawback of causing constant vomiting and a vicious circle being established. Granted that the secretions could pass quickly into the stomach and out through the other gastrojejunostomy opening, he feared it would have a bad effect in the way he had mentioned.

Mr. PATERSON, in reply, said that some of the points raised in the discussion were dealt with in his paper. In the time at his disposal he had thought it better to give a general summary of the paper rather than to read any part of it in detail, and so he had not in all cases given the reasons for the conclusions to which he had come. He had been much interested in Dr. Bolton's remarks with regard to the relationship between hyperacidity and the production of gastric ulcers, but he could not agree that the beneficial effects of gastrojejunostomy were due to earlier emptying of the stomach. While fully recognizing the value and importance of experimental work, he was not prepared to accept conclusions based on such work when they did not agree with careful clinical observation. He had made the observation frequently that the operation of gastrojejunostomy did not necessarily increase the motility of the stomach. He was convinced therefore that the beneficial effect of gastrojejunostomy on gastric ulcer was due not to hastening of the evacuation of the stomach, but to the resulting altered condition of the gastric contents. The effect of gastrojejunostomy was physiological and not merely mechanical. Dr. Bolton had objected to the proposal to divert the bile and pancreatic juice into the stomach, and had said that the administration of alkalis by the mouth was a preferable method of neutralizing the gastric contents. With the latter opinion he (Mr. Paterson) quite agreed. He had not suggested that diversion of the bile should be the routine treatment of jejunal ulcer, but a method to be adopted only after medical treatment and simpler surgical procedures had failed and the patient had suffered relapse. So far as he was aware, deliberate diversion of the bile into the stomach had not been tried in a case of jejunal ulcer, but he did not think that it would lead to constant vomiting, as Mr. Hutchinson anticipated. Regurgitant vomiting after gastrojejunostomy was due, not to the entrance of bile into the stomach, but to obstruction at the efferent opening which prevented the bile getting out again. The presence of bile in the stomach was not harmful provided the efferent opening was efficient. Dastre and Masse had shown conclusively that in dogs the presence of bile in the stomach did not interfere with the digestion and general health. The well-known case of rupture of the duodenum reported by Mr. Moynihan, as well as the results of the operation of cholecysto-gastrostomy in man furnished evidence to the same effect. He quite agreed with Mr. Spencer that jejunal ulcer was now less likely to follow gastrojejunostomy than was formerly the case, and, as he had indicated in his paper, he thought that this diminished liability was due to improved technique rather than to the adoption of a different type of operation. If the view that jejunal ulcer was due to the presence of free hydrochloric acid in the jejunum were correct, one would not expect it to occur after gastrojejunostomy for carcinoma, and it was presumptive evidence in favour of the correctness of this view that, notwithstanding the large number of times which gastrojejunostomy had been performed for cancer of the stomach, only one case of jejunal ulcer had been reported after operation for this disease, and in that case the ulceration was probably of infective origin. It was interesting to note that in several cases of jejunal ulcer, a jejunal fistula had formed spontaneously, and this appeared to be Nature's method of getting rid of the excess of acid.

Surgical Section.

June 8, 1909.

MR. J. WARRINGTON HAWARD, President of the Section, in the Chair.

Benign Tumours, Fibroma, Myoma and Lipoma encapsuled in the Wall of the Stomach.

By WALTER G. SPENCER, M.S.

THE connective-tissue tumours of the stomach are commonly sarcomata. Indeed, Fenwick, in "Cancer and Tumour of the Stomach," 1902, p. 350, stated that "We have not been able to find a single case in the whole of the literature where a large fibroid tumour of the gastric wall was above suspicion of malignancy." There are, however, exceptions to this general statement. A benign encapsuled growth, a fibroma, myoma or lipoma may grow to a considerable tumour, and yet can be removed without damage to the stomach. These benign growths have been mistaken for sarcomata, and an excessive operation has been done; when left alone, such tumours have caused death by pyloric obstruction, or by ultimately becoming malignant.

The case which I am about to describe is very similar to the tumour of the stomach described by Morgagni in his "Seats and Causes of Diseases," Epistle xix, c. 58. An old woman, aged 70, who had been a drunken monthly nurse, but had been incapacitated for eighteen months with loss of appetite, bronchitis and pleurisy, was examined after death. Morgagni found a globular tumour weighing about a pound situated in the posterior wall of the stomach about its middle. The tumour was covered by the muscular wall of the stomach, from which some hemispherical nodules of the tumour projected, and in which there were some large veins of the size of a goose-quill. The mucous membrane of the stomach was not involved nor adherent, but for an area of 2 sq. in.

corresponding to the tumour was rugose, yet quite healthy. The section of the tumour measured 4 in. by 3 in.; it resembled a scirrhus, being in some parts of almost bony hardness. It had apparently grown from the cellular tissue under the muscular coat. This tumour had been obscured during life by a fatty tumour in the subcutaneous tissue over the xiphoid cartilage and epigastrium.

The case I now describe was that of a hospital nurse, aged 46, who had suffered with abdominal troubles for some time, and had become so weak and anæmic as to be incapacitated for duty. The symptoms were partly referable to gastric trouble, and partly to the fact that the kidneys were movable. Dr. Miles, of Bewdley, sent her to see Mr. H. G. Barling, who discovered, in addition to the movable kidneys, a tumour in the epigastrium, which, he thought, lay in the gastrocolic omentum, and he advised the nurse to come back to the Westminster Hospital for operation. Mr. Barling, and also Dr. Miles, kindly wrote to me about the patient. At first I failed to profit by Mr. Barling's diagnosis. I found, on examination, that either kidney could be moved inwards to the middle line of the epigastrium behind the intestines. Neither kidney could be pressed downwards, as is usually the case, nor were they in any way floating. At the first operation, therefore, I made a right lumbar incision and examined and fixed the right kidney in position, and by putting my hand into the peritoneal cavity touched the left kidney and pushed it out into place. At the time I overlooked a third reniform tumour, or else confused it with the kidneys. As soon as I could, after the first operation, I again examined the abdomen, and then found there were still two reniform tumours, one the left kidney, the other lying over the promontory of the sacrum with its convexity directed downwards. This was the tumour Mr. Barling had discovered, but which I had previously overlooked; apparently its weight had caused it to sink down below the umbilicus. The patient then had pain, which she referred to the left kidney, and irregular vomiting; but the ward sister's observations tended to show that the symptoms occurred independently of whether the left kidney was in place or was to be felt in the epigastrium. I have no doubt that the pain was caused by the gastric tumour and was felt in the epigastrium, although the tumour had prolapsed below the umbilicus. She was also anæmic, hæmoglobin 44 per cent., which the vomiting may account for.

At a second operation I fixed the left kidney in the loin, felt the spleen in place, and then enlarging the left lumbar wound, I drew out the reniform tumour from over the promontory. At first sight it

appeared to be a perfectly normal stomach containing a soft body of the size and shape of a large kidney. For the moment I concluded that I had to deal with a hair-ball, because of the resemblance to a case which had been in the same ward under my late colleague, Percy Paton. However, on cutting into the stomach in its long axis, I found the cavity empty and free from inflammation and ulceration. The mucous membrane of the posterior wall was rugose, as in Morgagni's case, and pushed forwards, but moved freely over a solid elastic tumour. Having split the mucous membrane, I came upon the tumour, which was most easily shelled out without any hæmorrhage, only close to the lesser curvature were vessels seen which were clamped and tied. Suturing

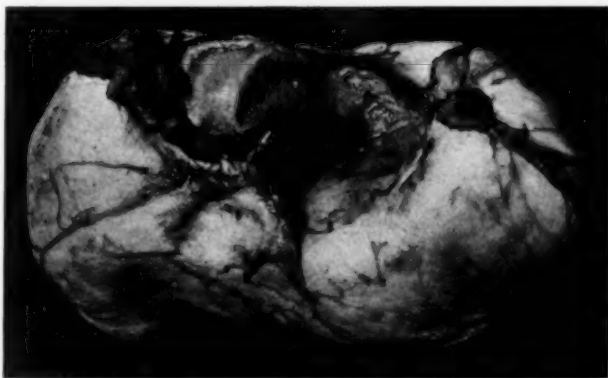


FIG. 1.

Submucous fibroma encapsuled in the wall of the stomach.

followed, and the patient recovered without incident. She has since got strong and regained a colour. Three and a half months after operation she was about to recommence nursing work.

The tumour has been mounted for the Museum of the Royal College of Surgeons. It has the shape and size of a large kidney (fig. 1), and weighed, after removal, 200 grm., or 7 oz. A section under the microscope shows it to be a dense fibroma.

Mr. S. Shattock has kindly furnished me with the histological description of the tumour as shown in the two microphotographs (figs. 2 and 3). Histologically the tumour consists of intersecting bundles of dense fibrous tissue furnished with scanty numbers of appertaining



FIG. 2.
Fibroma of stomach.

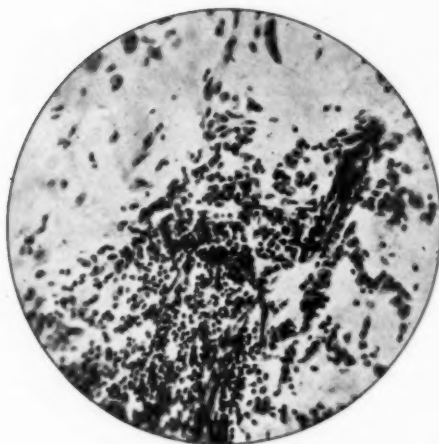


FIG. 3.
Lymphoid and plasma cells in section of fibroma of stomach.

lamella cells. Here and there a dense cluster of lymphocytes, accompanied with a certain number of plasma cells (Unna), is to be encountered; and both varieties of cell, moreover, are widely dispersed in small numbers throughout the section. Well-developed arteries and capillaries occur through the growth, but at somewhat wide intervals, the neoplasm presenting but little vascularity. The practical deduction from Mr. Shattock's description is, I take it, that the tumour contains cell elements which, at any time, might form the starting point of a sarcoma.

I show a museum specimen of a precisely similar tumour, only smaller, the size and shape of a small sausage. But it was placed in exactly the same position, with the convexity downwards, and the concavity close to the lesser curvature. It also, during life, formed a freely movable abdominal tumour which the surgeon took for an



FIG. 4.

Submucous fibroma of the œsophagus, removed post-mortem from a man, aged 28, who died of generalized staphylococcal infection arising from neglected boils.

intussusception, and cut out the tumour along with the middle part of the woman's stomach. Finding out what he had done, he had to reopen the abdomen to correct the suturing, but the patient died. The tumour is shown *in situ*, and is seen to be situated in the submucous layer, encapsuled, and separable from the mucous and muscular coats. Under the microscope it has the structure of a myoma or fibromyoma. I also exhibit a specimen of submucous fibroma from the œsophagus recently placed in the Westminster Hospital Museum, which shows very well the origin and position of such tumours (fig. 4).

Small tumours, myomas, fibromas, and lipomas have been frequently met with post-mortem. Kidd described a small submucous fibromyoma at the cardiac orifice of the stomach which had caused no symptoms during life. Cutler noted a myoma the size of a small bean projecting inwards from the anterior wall and growing from the submucous tissue.

In the Royal College of Surgeons Museum is a specimen, No. 2404 B, of a submucous fibromyoma of the wall of the stomach, the size of a pea, from a woman who had uterine fibromyomata. Murray, in a post-mortem on a man aged 64, found a submucous lipoma in the posterior wall of the stomach 2 in. from the pylorus. Even such small tumours, when they occur at the pylorus, may cause severe or fatal pyloric obstruction. Bénaky, of Smyrna, found, at the post-mortem of a man aged 65, that the pylorus was so blocked by a tumour that when the water-tap was connected with the cardiac orifice and the stomach distended with water, no fluid escaped by the pylorus. The tumour was a submucous lipoma, sessile, measuring 6 cm. by 3.5 cm. by 2 cm. It was easily shelled out, and weighed 38 gm. Pernices also met with a tumour the size of an egg in a man aged 75, which had blocked the pylorus and caused chronic dilatation of the stomach. Such tumours blocking the pylorus come early under notice, and may be successfully shelled out. Herhold removed a sessile myoma, the size of a hazel-nut, from the region of the pylorus, which it was tending to narrow. Poirier removed from the pylorus a tumour the size of a cherry; the symptoms, which had existed for a year, disappeared.

When, however, the benign character of such a tumour at the pylorus has not been recognized, and the symptoms of pyloric obstruction attributed to malignant disease, an excessive operation has been undertaken. Thus, in Sainter's case, a maidservant, aged 49, had a myoma the size of a bean, for which pylorotomy was done. But a year after the operation the patient still had so much gastric trouble following the operation as to be invalidated from work.

Von Hacker performed a partial gastrectomy on a girl, aged 24, for a fibromyoma growing from the lesser curvature of the stomach. The girl was well two years later, and married. Hahn did pylorotomy, and Bardeleben and Czerny gastro-enterostomy on the view that they had to do with sarcomas instead of benign encapsuled tumours.

If these tumours are left alone they may ultimately become malignant. Thus, in Brodowski's case, the tumour had been discovered eleven years before death, when the man was aged 46, but the tumour was taken to be a chronically enlarged spleen. When the man died, at

the age of 57, a myosarcoma was found weighing 12 lb.; the tumour had grown out from the greater curvature of the stomach into the layers of the omentum; there were secondary growths in the liver, and some free fluid in the peritoneal cavity. Part of the growth showed unstriped muscle fibres, part the structure of a fibrosarcoma undergoing myxomatous degeneration.

In Goebel's case, a woman, aged 69, died after exploratory laparotomy. At the post-mortem examination it was discovered that a myoma of the anterior wall of the stomach had ulcerated through the mucous membrane, and the tumour had broken down in its centre so that the centre of the tumour freely communicated with the cavity of the stomach. The tumour also projected from the anterior surface of the stomach to the size of a child's head, and had become covered by omental adhesions.

These tumours are not specially human in occurrence. In the Royal College of Surgeons Museum, specimen No. 2403, is a Hunterian specimen of the stomach of a codfish with a pedunculated fibroma 9 in. by 6 in. in diameter. Specimens 2404 and 379 are also specimens of submucous fibroma from a cod's stomach. Specimen 2404 (*a*) shows a fibroma 2 in. in diameter in the stomach of an eel. Petit described a myoma in the stomach of a horse measuring 5 cm. to 6 cm. in diameter projecting into the peritoneal cavity.

When these tumours grow from the anterior wall of the stomach they may project into the abdominal cavity, and, becoming pedunculated, sink down into the pelvis.

Perls Neelsen reported a myoma of the stomach weighing 6 kilos., which had dragged the stomach down into the pelvis. Ruprecht, in 1887, in the case of a man, aged 52, who for fourteen years had had gastric trouble, removed a myoma 10 cm. by 7 cm., weighing 251 grm., growing out from the subserous tissue of the cardiac end of the stomach; the man died on the eighteenth day of pneumonia, but the wound had been healing well. Von Erlach removed a tumour weighing 5.5 kilos. from the front wall of a woman's stomach, where it was attached by a peduncle, and the patient recovered. Herman, in operating on a woman, aged 60, for a supposed uterine fibroid, found a fibromyomatous tumour $4\frac{1}{2}$ in. in diameter, attached by a short pedicle $1\frac{1}{2}$ in. by 1 in. in diameter to the great curvature of the stomach from which it had grown. It was easily detached, and the suturing of the peritoneal surfaces stopped hæmorrhage. The patient was well two years afterwards. It was not a case of secondary attachment of an ovarian or uterine fibrous tumour.

Bircher described a very similar case. Fischer removed from the front wall of the stomach a fibrolipoma the size of a walnut; Halstead excised a pedunculated myoma growing from the lesser curvature the size of a small orange.

Although, therefore, it is impossible to diagnose such tumours except at the time of operation, and although sarcomas are more common than the benign tumours, yet benign tumours, either sessile and encapsuled, or pedunculated and easily detached, have been met with, whether fibromas, myomas, or lipomas. The removal of these tumours can be successfully accomplished without excising any portion of the stomach itself.

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DISCUSSION.

The PRESIDENT (Mr. Haward) said he was sure it was the wish of the Section to convey its best thanks to Mr. Spencer for his description of what was no doubt a rare disease. He supposed the moral of the case was that in dealing with tumours of the stomach one should bear in mind that they might be benign, and so perhaps avoid a needlessly extensive operation.

Mr. LEONARD BIDWELL congratulated the author on the case, and particularly on operating on a non-malignant tumour of the stomach; to those who did much stomach surgery the finding of an innocent growth was a delightful change. An interesting point was the possibility of such tumour being mistaken for movable kidney. Some stomach tumours presented such difficulties in diagnosis. A short time ago he was asked to stitch a movable kidney. He did not see the patient until just before the operation, and on examination he found that the kidney could be pushed a considerable distance across the middle line from the left side, and he believed the tumour was too movable to be a kidney, and so thought it better to explore by anterior incision. He made such an incision, and found it was a large tumour of the stomach which on removal proved to be malignant, although it had caused no stomach symptoms beforehand. The other point about which he wished to ask was, whether these tumours might really become malignant. He would like to know whether it was now recognized that fibromata did become malignant: because sarcomata were at times very slow-growing and for years caused no worse symptoms than fibromata. And it was rather against the idea of malignancy in the second case quoted that the tumour which had existed a long time broke down in its centre and ulcerated through without presenting signs of sarcoma.

Dr. MACNAUGHTON-JONES said that in myomata and myofibromata of the uterus there was no doubt of the occurrence of malignant degeneration, both sarcomatous and carcinomatous, and if it could occur in a tumour of the uterus, why not in one in the stomach?

The PRESIDENT, speaking on the question whether a benign tumour might become malignant, said he was inclined to think it was largely a matter of terms. He would have thought it was not so much a benign tumour becoming malignant as a tumour being invaded by malignant growth from without. Many years ago he published a case, which he watched for a long time, of an ordinary adenoma of the breast, which was eventually invaded by cancerous growth. He removed the breast, and both microscopically and clinically it was proved to be an adenoma invaded by cancer, because the patient had recurrence after a time. He supposed tissues of that kind, as other tissues, might be invaded by new growth; but he asked whether it was quite accurate to say that a tumour "degenerated" into a malignant growth.

Dr. MACNAUGHTON-JONES, in further comment, said he referred not to invasion of malignancy from without but to actual degeneration of the myoma, arising in the tissue of the tumour; and it was a matter of discussion in which tissue the degeneration actually began. In practically every present-day gynaecological text-book it would be found that sarcomata and carcinomata were given as degenerations.

Mr. SPENCER, in reply, said benign tumours of the stomach were rare, and that was the special point of the paper, that, in exploring, one might come upon them, and such benign tumours had had an excessive operation

done for them. He himself mistook this tumour for a movable kidney. The patient had three tumours moving about in her abdomen, and the two movable kidneys would not go down below their normal level, but either of them could be shifted into the epigastrium with great ease. And the gastric tumour had got between them, or he confused them; but Mr. Barling distinguished them, and moved the two kidneys back into their positions, and then felt a third tumour. He thought the question of benign tumours becoming malignant was one of terms. Surely from the naked-eye, surgical point of view they did see tumours which were called benign become surgically malignant. But when one came to transcendental or histological anatomy, he had shown a microphotograph, and Mr. Shattock's description of the cells which were found in places in the tumour. If those cells were seen alone under the microscope, they would do for those of sarcoma; and he connected that with the two histories which he had related, of long-standing tumours which apparently, clinically, ran a benign course, and ultimately became malignant. He thought that was the explanation in regard to tumours of the uterus such as Dr. Macnaughton-Jones mentioned. Strictly speaking, a tumour which in the end proved to be sarcoma, theoretically must have had sarcoma cells in it from the beginning. But from the naked-eye, surgical point of view those tumours were benign at one stage of their existence, although at a later stage they became malignant. The President's suggestion that there might be an invasion by cancer cells from without was another possible explanation.

Some Cases of Gastric Surgery.

By E. STANMORE BISHOP, F.R.C.S.

SURGERY, as applied to gastric disease, has of late years attracted much attention. The proposal to treat surgically gastric ulcer and its results is so (comparatively) recent that all records of cases so treated are valuable, whilst the results have been as a rule so satisfactory that many cases previously looked upon as incurable or neurotic have been subjected to at least exploratory operations with a view, if suitable, to more definite proceedings. These explorations have resulted in the discovery of some conditions which were to a certain extent unexpected, but which have proved amenable to surgical skill—such, for instance, as hour-glass stomach, or early carcinoma—whilst experience has shown that gastric or duodenal perforations are, if attended to without loss of time, by no means the hopeless accidents they were once considered.

Of complete gastrectomy I have had no experience.

Of partial gastrectomy there have been six cases. In all, operation was not sought until a well-marked mass was present which could be felt and in two cases seen. The lump varied in size from a celluloid ball to a tangerine orange. The patients were two males and four females; their ages ranged from 38 to 57. All the patients were in very poor condition and had become emaciated; probably this accounts in large measure for the mortality, four out of six. Two survived for a period of ten and twelve months respectively, but died ultimately, of certain recurrence in one case and exhaustion with probable recurrence in the other, but this was not verified post-mortem. The operation performed in each case was that of Billroth with end-to-end union, and this was followed in two cases by leakage at the angle between the vertical and circular sutures. In all cases great difficulty was experienced with the clamps (Lane's, Doyen's, Smith's) then in use (1902-6). As will later be seen, I have endeavoured to produce a more trustworthy appliance for this purpose. But since my last case, in 1906, I have not seen any in which this operation appeared justifiable. The later method of closing both openings completely and finishing by gastro-enterostomy evidently yields better results, and is the method which will probably be adopted generally in future. Moreover, as the

results of gastric surgery become better known we may hope to obtain such cases at a much earlier period. (This was written in April, since which time I have had two more cases which were treated in the latter manner; but, although with an immediate satisfactory result, they are of too recent date to be brought forward.)

Of the operation for gastro-enterostomy I have had forty examples, and my experience of this operation has been much more favourable. My mortality has been three: two from recurrent hæmorrhage in acute ulcer, and one from exhaustion three days later in a case of carcinoma. There has been no immediate mortality, and otherwise the results have been most gratifying. Of these cases, 18 were males and 22 females. Of gastric cases, 10 were males, 18 females; of duodenal ulcers, 8 males and 4 females.

The following are some of the more interesting cases operated upon by the writer in the Ancoats and Jewish Memorial Hospitals in Manchester during the last three years:—

Case I.—Ulcer of the pyloric area. Resultant contraction. Persistent vomiting for six months; great emaciation. Gastro-enterostomy. Recovery.

Miss G., aged 20, admitted to the Jewish Hospital under Dr. Graff, who courteously transferred her to me, August, 1907. This was a school teacher who had suffered for several years from epigastric pain after food and from flatulent distension. About the latter end of December, 1906, she began to vomit sour watery fluid intermittently, mingled with the food taken; this persisted up to the time of admission. For about the same time (her statements lacked precision) intermittent paroxysmal pain had been felt in the abdomen, which was not relieved by vomiting, but was, at first, by hot applications. This pain gradually increased, and later could only be controlled by hypodermic injections of morphia; it was equally severe by night as by day. In addition, food produced a sensation of weight which was relieved by rejection of the food taken. The bowels had been usually constipated, but there was occasional diarrhœa: there was no history of hæmatemesis. Amenorrhœa had been present for seven months. The girl was greatly emaciated and weighed only 6 st. She was in evident pain, and, though naturally of a very cheerful disposition, showed marked traces of previous and prolonged suffering. Her appetite was poor, and she dreaded taking food, as this immediately intensified her discomfort.

On inflation the lower border of the stomach was found to lie below the umbilicus; through the thin abdominal walls the marked peristalsis of the stomach could be seen culminating as a firm mass—the spasmodically contracted pylorus—on the right side; the upper part of the right rectus muscle showed tonic contraction, and the abdominal wall as a whole was retracted so that the costal margin projected high over the hollow abdomen; the iliac spines were also very prominent. There was marked hyperæsthesia over the entire epigastric region. After a test breakfast the stomach contents, removed one and a half hours later, showed total acidity 45, free hydrochloric acid, occult blood (benzidine test). There was no response to Murphy's test for biliary inflammation.

September 4, 1907: Posterior gastro-enterostomy was performed. The walls of the stomach were normal except near the pylorus on the anterior surface, where the peritoneal coat showed scarring. The pylorus was thickened but smooth and evenly enlarged; not specially hard and without nodulation. Very little chloroform sickness followed, and feeding by the mouth was begun on the third day. On the eleventh day there was some rejection of acid mucoid fluid. On the seventeenth day there was flatulent eructation, with vomiting the same evening of offensively smelling material; these offensive eructations lasted for over a month and from time to time the breath was offensive; the smell resembled that of pancreatic fluid. During the first fortnight she continued to lose weight, but during the third a gain was noted: the abdomen began to fill up and the pylorus to be less sensible to palpation; the pain after food had disappeared since the operation. She left the hospital on October 2, 1907.

On October 28 she reported herself. Her weight was then 8 st. $1\frac{1}{2}$ lb., so that she had gained over 2 st. She was quite free from pain at any time and was taking food well and without discomfort; she looked and said that she felt extremely well; there were still, however, occasional offensive eructations. About once a month she reported herself, each month steadily increasing in weight, and in July, 1908, her weight was over 10 st.; she was in excellent health, and very bright and happy; all eructations had completely disappeared for several months.

In this case the evidence of gastric ulcer was clear; and the improvement soon after operation very marked; it is noticeable that for some time, however, the eructation of foul-smelling gas continued, but without actual vomiting except once on the date mentioned above; this gradually but entirely disappeared. Slight gastric dilatation was already

present at the time of operation, but it was associated with increased motility. The loss of blood was slight, and could only be detected by the benzidene test.

Case II.—Recurrent gastric hæmorrhages. Posterior gastro-enterostomy. Repeated losses of blood after operation. Death from acute anæmia due to bleeding. Post-mortem finding of ulcer of posterior wall with close adhesion to pancreas and liver.

In December, 1907, I was requested to see a man aged 40, with Dr. Paget, of Manchester. For five years he had had an annual attack of hæmatemesis, from each of which he had pretty rapidly recovered under treatment. Just before our visit he had undergone two attacks within a fortnight of one another, and when I saw him the bed and floor of his room were covered with the coffee-coloured material he had vomited. His lips and conjunctivæ were white, his pulse thin and rapid (120); there was a great tendency to syncope, only prevented by a supine position with the head lower than the trunk. All food was immediately rejected, so that he had been fed for some days by nutrient enemata. With all possible care he was removed to hospital and posterior gastro-enterostomy was done. The pylorus was scarred, but no actual ulcer was found. After the operation he rallied well and began again to take and retain food by the mouth; at the end of the ninth day he became suddenly collapsed and extremely restless; no vomiting occurred, however, nor was there any melæna, for which the stools were carefully examined; the mucous membranes, which had partially regained their colour, again became blanched, and it was evident that blood was again being lost. From this state he again rallied and lived until the fourteenth day, when a fourth hæmorrhage took place and he died, apparently from sheer loss of blood. At the autopsy, a large ulcer was found on the posterior wall, just above the pylorus, firmly adherent to the pancreas and left lobe of the liver; during the separation of these structures the ulcer perforated. The new opening was soundly united and had apparently functionated well.

In this case the loss of blood had been and continued to be after operation very great and the interference was made soon after an attack of hæmatemesis; the position of the ulcer and its intimate attachment to the pancreas precluded any attempt to excise or invert it or to surround it by an occluding suture; one or other of which proceedings appear to be indicated in all cases in which it is practicable to carry them out. The case is indeed mainly serviceable as emphasizing the

conclusion already formulated by others, that the mere performance of gastro-enterostomy in cases where hæmorrhage is or has been actively present has no effect in preventing further loss of blood, as has been suggested on the ground that the ulcer is no longer subjected to tension during gastric peristalsis or to prolonged contact with irritating contents. Both these things are no doubt true and probably greatly assist in promoting cicatrization, but they are evidently not enough. Nor, on the other hand, does occlusion and inversion *per se* suffice to stop hæmorrhage from a gastric ulcer, as the following case shows.

Case III.—Perforation of stomach; operation within five hours; closure of perforation by suture; recovery from operation followed by repeated losses of blood by bowel; death.

T. D., male, aged 45, was admitted to Ancoats Hospital on January 12, 1909. This man was a carter in the employ of the Corporation. He had been exerting himself in loading a cart at 12.30 p.m. on the same day, and a few minutes afterwards, while at rest, a sudden intense pain was felt by him in the epigastrium, which brought him to the ground; he was brought without delay to the hospital. His last meal, which consisted of bread, ox tongue, and cocoa, had been taken at 8.30 a.m., about five hours before admission. After the attack he had been given some brandy, which rendered the pain more intense. He claimed to have been in good health until thirteen weeks previously, since which time there had been pain after food, commencing an hour after and lasting one and a half hours unless relieved by vomiting.

On admission he was a well-developed man without any sign of having lost flesh. The intense pain had passed away, but there still remained a general burning sensation diffused over the entire abdomen, the wall of which was generally rigid; this rigidity was most marked over the upper halves of the recti muscles, and perhaps a little more definitely on the left side; there was great tenderness on touch over the epigastrium and upper umbilical region, but none over McBurney's point, where indeed there was less rigidity than elsewhere. A diagnosis of perforation was made, and the abdomen was opened in the median line above the umbilicus, four hours after the occurrence of perforation. After division of the peritoneum the cavity was still closed by omentum, which was firmly adherent beneath and also required incision; as soon as this was done large quantities of greyish mucoid fluid rushed out. The sex and age of the patient had suggested a duodenal

lesion; therefore as soon as some of the fluid had been removed by sponges that portion of the gut was examined. It was difficult to see anything, for as soon as the soaked sponges were taken away, fresh and copious amounts of fluid took their place. As nothing could be found in that portion of the gut the stomach was drawn downwards and forwards; fluid gushed out more freely than ever, but it could then be seen that it was issuing from a rounded opening on the anterior wall of the stomach just below the smaller curvature, which until then had been hidden by the liver, against the under surface of which it had been pressed; no adhesions had been formed between the two organs. The opening was the size of a goose-quill, and, as in the next case, was the centre of a whitish patch, with the consistence of scar tissue fading away into the more supple tissues around; the opening was closed by two purse-string silk sutures superimposed. Three drainage tubes were placed, one through a stab wound in each loin, and one suprapubically in the pelvis; the first two of rubber, the latter of glass. The patient was placed in the Fowler position. No peritonitis ensued, but on the fifth day the stools contained blood, and this continued for four to five days; then there was an interval of nine days during which no further blood appeared; on January 31 a second appearance of blood was noted, which lasted for twenty-four hours, and a third on February 8, which lasted for a similar time. The drainage tubes were removed after forty-eight hours, and the suprapubic opening and that on the right side closed promptly, but the left-hand side opening continued to discharge. Eleven days later the temperature rose to 101.4° F., and on dilating this opening with sinus forceps a large amount of pus escaped; drainage was free, but the temperature continued to oscillate between 100° F. and 101° F. Part of the tenth rib was removed in order to provide a wider opening, but the amount of pus steadily diminished without any perceptible lowering of the temperature; no fresh collection could be found, and the discharges from the wound were found to be sterile when incubated upon agar plates. Bleeding again recurred on March 6 and March 7, blood appearing in quantity in the stools, and the patient becoming exsanguine. He died on March 8.

The combination of both proceedings gives better results, but such a combination takes up more time than can always safely be spent after perforation has occurred, and it is satisfactory that occlusion is the most efficient, as in every case that has to be done if only to prevent further effusion of gastric contents; the anastomosis can be effected later when the patient has sufficiently recovered.

As an example of the operation in two stages for perforation the following case may be cited.

Case IV.—Perforation of duodenum. Enterorrhaphy. Fourteen days later gastro-enterostomy. Recovery.

L. I., aged 19, female, walked into the out-patient department of Ancoats Hospital at 5 p.m., on January 28, 1909. At 4 p.m., on the same day, she had been attacked by a violent pain in the epigastrium; after some minutes it had sunk to the level of the umbilicus, at the same time becoming much less intense. The history she gave was that for several months she had suffered from pain of a much milder type in the epigastrium, which came on one hour after food and was relieved by vomiting. To this statement she adhered after the operation, which disclosed a condition which did not appear to fit this anamnesis. There had been anorexia and some loss of flesh; she had never vomited blood. Both recti muscles were found to be rigid in their upper halves, that on the (patient's) right being rather more unyielding than the left; the abdomen generally was tender, but the pain was much less and was decreasing. Immediate operation was decided upon. At 8 p.m., four hours after the appearance of acute pain, the abdomen was opened in the median line above the umbilicus; all the veins in the abdominal wall were engorged, and the blood which escaped from the divided vessels appeared to be darker than usual.

On opening the peritoneum no gas or fluid escaped; the anterior wall of the stomach was reddened and a few stray flakes of lymph were seen. On pulling the stomach forward a minute opening was seen in the antero-superior wall of the duodenum, about $\frac{1}{2}$ in. distal to the pylorus, from which a thin whitish fluid spurted out. The opening was in the centre of a whitish thickened patch which faded away into the surrounding wall of both stomach and duodenum. It was closed by two superimposed purse-string silk sutures. Very little free fluid was found, but all the surfaces around were greasy to touch. One rubber drainage tube was placed in the right loin through a stab wound; this was removed forty-eight hours later, very little fluid having escaped by it. As the patient bore the anæsthetic badly nothing further was done at this time. A fortnight later, everything being healed and the patient in good condition, a posterior no-loop anastomosis was made; from this also she recovered perfectly and left the hospital cured on March 12 without any pain, vomiting, or discomfort during digestion.

When the abdomen was opened for the second operation an opportunity was thereby afforded for examination of the area originally affected. The site of the perforation was firmly adherent to the fundus of the gall-bladder, which could, however, be wiped away with gauze, disclosing a small closed spot with but little evidence of the puckering around produced by the purse-string sutures, both of which had disappeared.

That such gastric or duodenal ulcers do sometimes heal spontaneously under favourable conditions has been well known for many years, but that this result is not always accompanied by a return to normal health is also a matter of experience and is exemplified by the following case.

Case V.—Three years' history of pain and vomiting after food. Clear history of perforation one year before, confirmed by direct observation during operation. Recovery from perforation under medical treatment, but persistence of symptoms. Posterior gastro-enterostomy. Perfect recovery.

J. R., male, aged 59, admitted to Ancoats Hospital, October 28, 1907. This man had complained for two or three years of pain in the epigastrium coming on two hours after food; for the same time there had been intermittent attacks of vomiting which relieved him, and towards the end of the act on several occasions material like coffee grounds had been brought up. Twelve months before admission, on return to work after dinner in the middle of the day, he was suddenly seized by intense pain in the epigastrium which totally incapacitated him. He was carried home, and remained in a collapsed condition for some time. He was treated medically and slowly recovered. At the time of examination he said that usually he felt well in the mornings before breakfast; for this meal he had no inclination, but he could eat dinner. Two hours after dinner a feeling of distension came on, which lasted all the afternoon. About two and a half hours after dinner a dull, aching pain was felt in the epigastrium which was relieved if he could vomit, as he frequently did.

On November 1, 1907, the abdomen was opened. To an area of the anterior stomach wall about 2 in. in diameter, which was thickened and scarred, the omentum was very firmly adherent. Gastro-enterostomy—no loop, posterior—was performed with a double layer of continuous sutures. Recovery was prompt; all pain and vomiting ceased at once; soft food could be taken and digested forty-eight hours later, and, from being somewhat emaciated, he began to put on weight, gaining 3 st. in the following two and a half months. When seen again in February,

1909, fifteen months after operation, he said that he had been at work as a painter, often on scaffolding high above the ground, since January, 1908, and had not been absent from work for a single day. He could eat anything with enjoyment, and without any sequent pain, vomiting, or eructations of gas, whilst the bowels had always acted perfectly.



FIG. 1.

Duodenal ulcer. Gastro-enterostomy. Shows closure of stoma after filling of intestines, the stomach still retaining some food. *a*, stomach; *b*, clear space between stoma and food contained in attached intestine; *c*, intestine.

The rate and completeness of recovery appear to differ greatly in different persons; thus in the first case mentioned but little improvement was seen during the first fortnight, although in the end the result² was

all that could have been wished; the patient began to put on weight after the third week, but for months there were frequent and annoying eructations of foul-smelling gas, though these ultimately and entirely disappeared. In another case, in which the stomach was much dilated, although pain quickly disappeared, general improvement did not show itself for five months, but then became steady and marked. In the last, and one may fairly say in the majority of cases treated by the posterior no-loop operation, the improvement was rapid, whilst eructation, vomiting, and pain were not seen at all from the time of operation. Occasionally the usual well-known symptoms of gastric ulcer are associated with nerve conditions also apparently relieved by gastro-enterostomy, though it is difficult to understand in what way their elimination is brought about. The following case is a good instance of this.

Case VI.—Two years' history of pain occurring two hours after food, and located in the epigastrium and right hypochondrium, relieved by vomiting. Seven years previously an attack of gastric hæmorrhage. No blood in vomitus, but (occult) blood in fæces. Persistent acute pain in right arm following the course of the ulnar nerve from shoulder to fingers. Disappearance after gastro-enterostomy.

B. H., female, aged 31, admitted to private hospital, January 6, 1905. For some years she had complained of pain occurring about two hours after food; this pain was situated in the epigastrium and right hypochondrium; there had been intermittent vomiting which always relieved the pain; this vomiting was becoming more frequent and was peculiarly sudden. Hyperæsthesia was marked in the epigastric region spreading downwards and to the right along the costal margin; the epigastric pain was increased by pressure. There was a history of hæmatemesis seven years previously, but no blood could now be found on examination of the vomitus; occult blood was, however, found on three occasions in the fæces with the benzidine test. Associated with this there was pain along the entire course of the ulnar nerve from the shoulder to the fingers of the right arm; this was persistent and continuous and had been present for over six months; yet there was no herpetic eruption over the nerve or any atrophic change in the parts supplied. Pressure over the course of the nerve appeared to intensify the pain and the line of distribution was mapped out with fair accuracy.

On January 8 posterior gastro-enterostomy was done. There was some whitish discoloration over the first part of the duodenum, but the pylorus was normal and the stomach not dilated. She made a good

recovery, all vomiting and gastric pain disappearing; at the same time the pain in the arm vanished and there has been no return of either up to the present date (March, 1909).

The operation of gastro-enterostomy is usually performed for cases of gastric or duodenal ulcer, but is also capable of yielding excellent results in gastric carcinoma where excision is impracticable, as in the following case.

Case VII.—Gastric carcinoma, affecting body and entire smaller curvature; feeble condition of patient. Posterior anastomosis with jejunum. Recovery with greatly improved appetite and loss of all pain during digestion; increased weight.

Mrs. H., aged 65, admitted to private hospital, February 10, 1909; sent by Dr. E. E. Smith, of Hyde. This patient had complained of some discomfort beneath the ribs on the left side for four years, more or less increased by food. (No definite details were obtainable.) Fifteen months before admission she had noticed a small hard mass on the right side beneath the costal margin, but had not paid much attention to it as it was not painful. For the last nine months, however, steadily increasing pain had been felt after food, which latterly had become intolerable; so intense was it that she was not able to sleep. She noticed that the pain was always worst at night; food induced acute exacerbations which made her roll on the floor in a fruitless endeavour to obtain relief. For some six weeks she had required hypodermic injections of morphia. She did not vomit much or often, but had had waterbrash several times: she had never brought up blood. For the last four months there had been notable loss of weight. The bowels were constipated, and the stools were said to be ribbon-shaped: defæcation was said to increase the pain. On the right side, midway between the costal margin and the umbilical plane, a nodular hard mass could be felt which was movable laterally, slightly tender to touch, and about the size of a walnut. Manipulation of this mass started visible peristalsis, which excited the pain. A provisional diagnosis of carcinoma was made, but its location appeared more likely to be the transverse colon than the stomach.

On February 17, 1909, an exploratory median incision was made above the umbilicus; the transverse colon proved to be free, but the pyloric vestibule was the seat of a cancerous mass, several secondary nodules being scattered over the body of the stomach, and numerous hard glands could be felt in both the upper part of the great omentum,

and especially in the gastro-hepatic omentum along the lesser curvature as high up as the cardiac opening.

It was evident that removal of the major part of the stomach would be necessary if any attempt at radical cure was to be made. The condition of the patient and the fact that anaesthetization was very badly borne appeared to negative this; posterior gastro-enterostomy was therefore done, the jejunum being united to a clear area at the fundus of the stomach, and the abdomen closed. Even this was evidently almost too much, and shock was, for a few hours, pretty well marked. After recovery from this condition she became very restless, and complained of much pain, chiefly in the back between the shoulders; morphia and atropine, however, relieved her, and after the sleep thus induced she awoke much easier. This improvement continued, and, having regard to her age, she was encouraged to get out of bed on the eighth day. She found that the upright position was the most comfortable during the earlier days, though within a fortnight she was comparatively free from pain in every position, and this helped to induce her to get out of bed quickly. By the end of a month she was able to return home, having slightly increased in weight, being able to take and digest food with comfort, and sleeping for four to five hours every night. Later, pain of a dull aching kind was complained of, mainly across the spine about the level of the twelfth dorsal vertebra, evidently due to the presence of the carcinoma; but this was not increased by food, and was very much less than before the operation.

On March 10, in reply to a letter of mine, regretting that no more could be done for his patient, Dr. Smith writes: "I am quite satisfied with the result; it is nice to feel that there is very decided relief. One feels quite justified for having persuaded her to submit to the operation."

Instances of hour-glass or bilocular stomach are not very frequent, but Veyrassart has collected a series of seventy-six treated by gastro-enterostomy: three only were treated by a combination of gastro-gastrostomy and gastro-enterostomy, two by Moynihan and one by Hochenegg; all recovered. I have one example of the same kind to record.

These cases present a very definite problem to the surgeon, as to the solution of which there is room for much difference of opinion. Veyrassart lays great stress upon the necessity of uniting the intestine with the cardiac pouch, as if done with the pyloric pouch, and the operation cut short, the result will not be good. Twenty-eight operations have

been recorded by Pinatelli, in five of which the pyloric pouch was utilized, with four deaths. In Veyrassart's list the pouch is only specified in twenty-one; in thirteen of these the cardiac pouch was utilized, without death; in eight the pyloric pouch, with six deaths. Guinard reports a death from this cause. Bier, Moynihan, Sidney Martin, Bilton Pollard and Hartmann have all had cases in which the attachment of intestine to the pyloric pouch has been followed by a fatal result. In some of these, as in that of Guinard, the pyloric segment has been so large as to appear to constitute the entire organ, the small cardiac pouch being only discovered post-mortem. In all cases it is evident that a very careful search must be made for this possibility before the anastomosis is made, and if gastro-enterostomy is the only operation performed the opening must be made into the cardiac pouch. When, however, it is combined with gastro-gastrostomy, which eliminates the division between the two pouches, it would seem more reasonable to drain the most dependent, the pyloric pouch. Of the two operations, however, the gastro-enterostomy is undoubtedly the most essential, and, as it is impossible beforehand to feel certain that a patient requiring both will be able to stand the prolonged anæsthesia and manipulation which the combination demands, the anastomosis will naturally be done first; and as this may be all that can be done, it follows that it will be done with the cardiac pouch, so that the decision would seem to be already made by the circumstances inevitably present in such cases leaving no alternative. The theoretical advantages which might be found by a combination of gastro-gastrostomy with pyloric gastro-jejunostomy would seem doomed to remain theoretical only. Certain surgeons have attempted to drain both pouches, and the methods of Clément and Monprofit are intended to effect this object; both, however, are tedious, and would appear to be open to the risks of internal hernia later, between the two attachments.

In the *Revue de Chirurgie*, March, 1909, Delore and Alamartine, in an exhaustive paper on this subject, advocate strongly removal of the median portion of the stomach, including the constricted area which in the three cases they report, as also in eleven cases reported by Riedel and quoted by them, was caused by callous but still active ulceration. In those cases the pathological findings left it certainly an open question whether the surgeon had to do with an ulcerated cancer or with an ulcer on which cancerous changes were engrafted, but in either case showing urgent need for entire removal. The case I have to report is scarcely parallel; even if, as would seem probable, the condition was initiated by the contraction of an ulcer, at the time of operation that

ulcer had entirely disappeared and had left no trace of its presence, neither external scarring, thickening of tissues, nor internal lesion being discoverable; there was, therefore, no urgent necessity of that kind to influence the operative decision.

Case VIII.—Bilocular stomach. Gastro-enterostomy with the cardiac pouch. Gastro-gastrostomy; cure.

M. C., aged 32, sent by Dr. Davie, of Fallowfield, February, 1909. For more than nine years she had suffered from attacks of epigastric pain and vomiting; after some months of this she had a sudden attack of very acute pain, which was followed by an illness which confined her to bed for fourteen weeks, and was diagnosed as "inflammation of the bowels"; from this she slowly recovered and returned to her duties in the post office. During the ensuing years she had several minor attacks of vomiting, sometimes with pain, sometimes without; these attacks frequently came on about 2 o'clock in the morning, and had no connexion with the menstrual periods, which were regular and painless. Latterly the attacks had become more frequent and severe, whilst she had lost weight; a year previously her weight was 9 st. 7 lb., at the time of admission it was 8 st. 2 lb.

The advantages of X-ray examination, so well described by my friend, Dr. Barclay, at the January meeting of the Electro-Therapeutical Section of this Society, were very evident in this case; indeed, it cleared up completely what before was a somewhat puzzling condition.

At the time of examination there was but little definite evidence, and what there was was conflicting; there was the history of persistent vomiting, but the contents of the stomach on analysis proved to be normal; there was no occult blood, no hyperhydrochloria, no sign of fermentation; some little hyperæsthesia was noted posteriorly on the left side on one occasion, but on a second examination this could not be found; neither Wölffler's first or second sign was present; Von Eiselberg's and Moynihan's tests with seidlitz powders were not applied, because in one of my previous cases of gastric ulcer very serious, and for the moment apparently dangerous, symptoms followed the sudden distension with CO₂. Prolonged, violent, and excessive vomiting, with the ejection of quantities of blood, was the result in that instance, and ever since I have used such tests with great reluctance. The present patient was seen by other members of the staff, and the general opinion, including my own, was that it was a case of neurosis, but the use of the

fluorescent screen quickly demonstrated the actual condition. The accompanying photographs were taken. In the first (fig. 2), a small stomach is seen ending somewhat abruptly at a higher level than usual; the visible portion was in a state of violent peristalsis, which produced



FIG. 2

Bilocular stomach. Shows small cardiac pouch (a).

marked contractions at the lower end; some of these can be seen in the photograph. In the second plate (fig. 3), taken ten minutes later, the main mass of food can be seen lying nearly as low down as the cæcum, whilst a thin line unites the two collections.

At the operation, precisely this configuration was found. The stomach, far more than any other I have seen, resembled a length of large intestine with a well-marked constriction near the centre; at this point the stomach was contracted until the calibre would barely admit two fingers.



FIG. 3.

Same case, ten minutes later. Shows large pyloric pouch connected by narrow tube with cardiac pouch (view from behind).

The upper border corresponding to the lesser curvature was not affected, the greater curvature only was implicated; this was drawn gradually upwards so that the pouches (cardiac and pyloric) communicated by a narrow channel at their upper extremities. Although the history

pointed to cicatrization of an ulcer as the producing cause, no trace of such an ulcer, or of its scar, could be found; the external surface was perfectly smooth and of normal colour, nor when the stomach was opened in this area could anything abnormal be seen in the mucosa. As it was doubtful if the patient could stand a combined operation, posterior



FIG. 4.

Same case, four months after gastro-gastrostomy and gastro-enterostomy. Shows nearly normal, though large, stomach.

gastro-enterostomy was first done between the cardiac pouch and the jejunum; when this was finished, as her condition remained good, and she was taking the anæsthetic well, gastro-gastrostomy was performed, uniting the two pouches by an inverted U-shaped incision after the

method of Kammerer, which restored the normal shape of the stomach.

The patient recovered without any unfavourable symptom, and returned to her home on March 7, having had no pain or tendency to vomit. But the after-history of the case renders it, perhaps, a more interesting subject for discussion. Whilst she remained in the hospital, and more or less in the recumbent position, progress was rapid and uneventful; her food was taken with appetite and digested with ease, whilst her general condition rapidly improved. But a week after her return home she began again to complain of dull pain and a sensation of weight and dragging referred to the site of the pyloric pouch, with occasional vomiting. X-ray examination showed the bismuthized food collecting in the lower pouch; this had evidently passed over the new stoma without escaping through it. If these symptoms persist, I intend later to reopen the abdomen and to excise the pyloric pouch, closing the stomach above and the duodenum below, as Alamartine, Delore and Mayo advise, trusting to the anastomosis already made. On this point, however, I look forward with interest to any discussion which may follow. (This was written in April; in May, matters began again to improve, and on the 31st of that month the accompanying photograph (fig. 4) was taken, showing a great improvement in the size and shape of the stomach, but some defect in motility, peristalsis being sluggish and wanting in force. The general condition was much more satisfactory, fully justifying the policy of delay.)

All these operations, with the exception of one done in 1902, have been the posterior no-loop operation of Mayo and Moynihan. I have not seen any vicious circle vomiting afterwards in any of my forty cases. The technique of the operation is now practically settled, and is familiar to all surgeons; one point, however, first described by Mayo, may be worth referring to. That writer drew attention to a triangular fold of peritoneum in certain cases descending from the duodeno-jejunal opening and losing itself upon the jejunum; he advised its removal before anastomosis was performed. It will also be in the memory of all abdominal surgeons that a discussion arose some months back as to the correct line of apposition between the two viscera. May it not be that this fold indicates the proper angle at which the united jejunum should lie, and that, so far from removal, it should itself be retained as an additional means of strengthening and supporting the line of union? In two of my cases it was observed and was used in this way. The result

was good, and in the later cases the angle so indicated has been followed as nearly as could be done.

Another point to which I have as yet seen no reference is the peculiar naked-eye appearance of the first 7 in. or 8 in. of the jejunum; this coil, which is the one desired for anastomosis, looks as if it had been sodden; it has not the polished surface of the rest of the small intestine and is covered by a number of minute eminences. There is besides a curiously "moulded" appearance as if the gut contained putty which had been irregularly compressed. Of course, the surgeon relies mainly upon the fact of its emergence from the duodeno-jejunal opening, upon its anatomical position in fact, to assure himself that it is the coil required, but this is a confirmation which supplies additional certitude, and is therefore welcome. I am not sure that this is always the case, but I have seen it in so many that I should confess to a feeling of doubt and uneasiness if it were not recognizable.

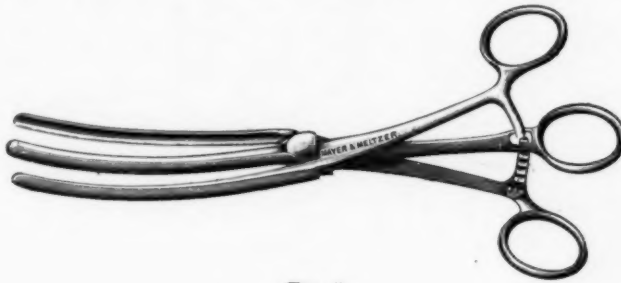


FIG. 5.

For approximation of the two viscera, and for ensuring their constant relative position during the act of suturing, a three-bladed forceps has proved extremely useful; the original model is a German one, which I have modified so as to make it lighter and more effective (fig. 5). Mr. C. J. Bond considers that the use of any clamps increases the tendency to the eversion of the mucosa and produces stagnation of the circulation in the compressed tissues, thus decreasing the prospect of a satisfactory union. He therefore prefers that the viscera be held together by the fingers of an assistant during the work. I have not found these conditions, which undoubtedly exist, to have any serious effect, and the additional security against escape of contents during the process of suturing, with the certainty of retaining the relative positions of the viscera throughout, appear to more than compensate for them.

In the operation of partial gastrectomy the want of fitting suitable clamps is most evident. Many surgeons appear to use clamps originally designed for operations upon intestine, and these fail when brought to bear upon the stomach walls. I have seen a Doyen's clamp snap across with the strain, permitting the contents of the stomach to escape into the peritoneum, with a fatal result. But the more usual mishap is due to the crossing of the blades, so preventing due apposition throughout their whole length. Moynihan's clamp, heavy as it is, has this defect. To ensure the meeting of the tips, Kocher has designed a clamp with a cross-bar attached to one blade in which are openings which fit over a blunt spike at the end of the other; it must, however, be obvious that the distance of these openings from each other will determine the amount of pressure produced irrespective of the wishes of the operator.



FIG. 6.

Moreover, the fixing of this cross-bar is a matter of some difficulty in the position in which it has to be used and it is awkward and clumsy in action.

The walls of the living stomach are different from those of any other organ in the body; they are muscular and tend to retract forcibly when divided, much more forcibly than those of the intestine, which most closely resemble them. They are lined by a slimy membrane and when two such surfaces are held together they tend to slide over one another; such walls are therefore peculiarly difficult to maintain in definite contact; slowly and insidiously the one ceases to lie exactly in the same relation to the other, gradually they slide past each other until one is free in the centre, and of course all control over the fluids within is immediately lost. The instruments shown (fig. 6) are specially constructed for this work; the large eye of the upper blade fits over the bent probe-like

extremity of the lower so as to ensure both the parallelism of the blades, and to prevent the escape upwards of the organ compressed; an occurrence frequently noticed when instruments acting upon a central hinge are used. The strong steel pins in the upper blade after penetrating both coats of the stomach fit into openings in the lower blade, and thereby effectively prevent any sliding of one coat over the other when both are divided. These instruments are made in pairs, so that these pins are in each case nearest to the line of incision. When they are firmly closed it is impossible for the contents to escape even if the stomach is cut fairly close to their edges. As already stated, I have recently had two more cases of partial gastrectomy, so that these clamps, which had previously been tested upon the stomachs of animals and upon the cadaver, have now been also tested upon the living human stomach and have amply proved their efficiency. These instruments, as well as the three-bladed clamp, have been made for me by Messrs. Mayer and Meltzer, of Great Portland Street.

I have to thank my colleagues of the Ancoats and Jewish Hospitals, especially Dr. Craven Moore and Dr. Graff, for their courtesy in sending me cases, and Dr. Bythell and Dr. Barclay (chiefs of the radiographic department at Ancoats) for the many valuable photographs and drawings with which they have supplied me, some few of which I have shown to-night.

DISCUSSION.

Mr. LEONARD BIDWELL said the paper traversed a great deal of ground, and he would only refer to one or two points. With regard to partial gastrectomy, he was glad to hear that Mr. Bishop now performed gastrectomy by closing the two ends and doing a gastro-enterostomy. The only difference in the procedure which he (Mr. Bidwell) suggested was that he would find it easier to do a gastro-enterostomy before removing the growth. The best procedure was to divide the stomach near the pylorus, and detach the gastro-hepatic omentum and the great omentum, then one could pull the stomach up, exposing the posterior surface of the cardiac end of the stomach, and it would be found easy to do the gastro-enterostomy before the tumour was removed. He congratulated the author on the result of his gastro-enterostomies, as malignant cases as well as those of ulcer were included. He thought results published giving about 1 per cent. mortality for gastric ulcer were misleading, and Mr. Bishop's forty cases with only three deaths was a very satisfactory result. He (Mr. Bidwell) was able to relate a case of hour-glass stomach which was treated by double gastro-enterostomy. The case was sent to him as one

of ovarian tumour, which showed how great was the dilatation of the pyloric part of the stomach. There was a separation of 2 in. between the gastric and pyloric portions of the stomach, and between the pyloric portion and the duodenum was a narrow channel about $1\frac{1}{2}$ in. long, so that if he had simply done a gastro-enterostomy in the gastric portion of the stomach, he would have left the pyloric portion, a large cystic swelling, undrained. It was therefore essential to drain it into the jejunum. He took precautions against strangulation in the loop between the gastro-enterostomies by suturing the jejunum with a few interrupted sutures to both gastric and pyloric portions of the stomach. There was practically no part left free. The result was absolutely satisfactory. He never used intestinal clamps, as he always felt that they might damage the vitality of the parts, and he felt no dread of the escape of stomach contents. Moreover, stomach contents were not really very irritating, especially as they only touched the outer surface of the stomach or jejunum, and there was no chance of their getting inside the peritoneum, because if one operated with a fairly small incision, the incision was completely plugged up by protruded intestine and stomach. Therefore he regarded the question of the escape of stomach contents as rather a bugbear. He kept stomach and jejunum in apposition by sutures passed as guides, and he avoided the use of clamps. Mr. Bishop's clamps were very strong and likely to prevent slipping, but he (the speaker) would not like to put those formidable teeth through a stomach with which he was dealing. The author said he had not seen a vicious circle after the posterior operation. No doubt a vicious circle would occur after the posterior operation when a loop was left between the first part of the jejunum and the anastomosis. He thought every surgeon would agree that a gastro-enterostomy which was done in a case where there was no stricture of the pylorus was only of temporary avail; that as soon as the spasm of the pylorus subsided the food would pass through the pylorus again, and the gastro-enterostomy would be practically useless. Therefore, in operating for duodenal ulcer, he thought it could not be said that a cure was obtained by gastro-enterostomy unless steps were taken to occlude the pylorus, because the discharge of stomach contents over the cicatrized duodenal ulcer was liable to be followed by a return of the ulcer. He had had two cases of duodenal ulcer which had recurred with severe hæmorrhage a couple of years after gastro-enterostomy; and since that time he had always occluded the pylorus at the same time as he did the gastro-enterostomy.

Mr. CHILDE congratulated Mr. Bishop on the selection of cases which he had brought forward, as any surgeon who had done much gastric surgery would at once recognize that they were all instructive cases, and typical of the conditions met with. He had operated upon the stomach between eighty and ninety times during the last ten years, and had had examples of almost all the cases Mr. Bishop had dealt with. He had had three cases of hour-glass stomach. In one of them the distal pouch was small and was neglected, and no inconvenience resulted. In another there were two large pouches, and a double gastro-enterostomy was done, and with a perfect result; the patient was

as well off as after simple gastro-enterostomy for an ordinary case of pyloric obstruction. The third case occurred in the first operation on the stomach he performed; there was a very long history, and the patient had a lump at the pylorus which was palpable; he opened the abdomen with the view of doing gastro-enterostomy, found a dilated stomach, and performed the anastomosis, but the patient was not relieved and died shortly afterwards; the post-mortem examination showed a pyloric ulcer, a dilated distal pouch to which anastomosis had been performed, and beneath the ribs was an enormous cardiac pouch, which had been missed at the time of the operation. He published it at the time as a warning, and there were other published cases in which a similar mistake had been made. A similar error was not likely to occur to him again, and it was unfortunate that it should have been his first stomach case, but under such circumstances it might easily occur to anybody. With regard to perforation, he thought all surgeons must gratefully recognize that general practitioners were becoming alert to the fact that immediate operation was an urgent necessity, and his experience during the last two years had been very happy in this respect. He had been summoned and performed operations within two hours of perforation, and when that was the case one could almost ensure a good result, and a gastro-enterostomy could be performed at the time that the perforation was closed, as he had done in his last five cases. One medical man, upon whom he operated last year, perforated while in his motor car at 2 o'clock, and by 4 o'clock the perforation had been closed and gastro-enterostomy done. If the patient was not seen until some time after perforation it would often be impossible to do the double operation. He had some time ago invented a clamp, which he had not published because a traveller walked into his consulting room with a similar clamp which had been made abroad. It enabled one to adjust the pressure of the blades exactly as desired.

Mr. W. G. SPENCER asked Mr. Bishop to explain his line of incision into the jejunum in relation to the fold shown on the skiagram. With regard to the question of using clamps, he thought two sorts of clamps were required—one for crushing thick portions of stomach and intestines, and a fine clamp to be applied along the line thinned by crushing. If one had a strong clamp, and so large as to meet the objections of Mr. Bishop, it would be so much in the way for the rest of the suturing. A fine clamp was needed when tucking in and suturing up.

Mr. STANMORE BISHOP, in reply, thanked members for discussing what he felt was a very incomplete paper. He proposed to try Mr. Bidwell's suggestion of doing gastro-enterostomy before removing the portion of stomach. He had not done it up to the present time, because he had been afraid he might find it necessary to remove more of the stomach than at first seemed likely, and then the gastro-enterostomy might be in the way. Obviously in certain cases it would be very useful. He asked Mr. Bidwell how long it was since the double gastro-enterostomy was done, because the adhesions between the peritoneum tended to separate after a time. One might put stitches into the peritoneum,

and interrupted sutures uniting the structures in the peritoneum, coated with that membrane, and in two years afterwards those surfaces would be free and loose. He gathered that the speaker merely put sutures through the peritoneal surfaces opposed. No doubt there was a sufficient union at first, but the ultimate result was as he had said. He asked whether his case had been done long enough ago to eliminate the possibility of that danger. The objection to the teeth in the clamp which he showed was much a matter of opinion. He asked whether Mr. Bidwell could consistently object to the teeth in the clamp, the points of which were inverted into the re-united stomach, when he used needles with absolutely no anxiety in putting in sutures so as to invert the stomach. He also wondered how Mr. Bidwell occluded the duodenum. One could always get a temporary occlusion at the pyloric portion by putting sutures round it and stitching through it. It had been shown in the days of Travers that all sutures passed into the intestine gradually worked their way into the intestine, and were discharged through the lumen of it, and the intestine regained its normal calibre, so that the occlusion could only be temporary, unless one excised a portion of the pylorus or of the intestine and closed the ends so that they were absolutely separated. With regard to the position of the incision in the jejunum, it was behind that fold. If one opened the gastro-colic omentum and brought out the stomach through it, it would lie behind that fold. The fold was attached to the line of suture and came down over it, and shielded it in the same way that one shielded the same line when one fixed the gastro-colic mesentery around the edges of the occlusion which had been already made.

PROCEEDINGS
OF THE
ROYAL SOCIETY OF MEDICINE

VOLUME THE SECOND

COMPRISING THE REPORT OF THE PROCEEDINGS FOR THE
SESSION 1908-9

THERAPEUTICAL & PHARMACOLOGICAL SECTION



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1909

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The Council think it right to state that the Society does not hold itself in any way responsible for the statements made or the views put forward in the various papers.

Therapeutical and Pharmacological Section.

October 6, 1908.

Dr. T. E. BURTON BROWN, C.I.E., President of the Section, in the Chair.

The British Pharmacopœia: its Scope and Object.

By NESTOR TIRARD, M.D.

It is ten years since the publication of the present British Pharmacopœia, and ever since 1898 there have been many indications that active steps were being taken in preparation of a new issue. The reports of the General Medical Council from 1898 onwards have recorded almost continuous work connected with the Pharmacopœia. Researches have been conducted in the laboratory of the Pharmaceutical Society, and by numerous workers selected by the General Medical Council; the published criticisms of the volume have been collated, and many have formed the subject of fresh investigations. But while all this work has been steadily pressed forward by the Council with the cheerful coöperation of the Pharmaceutical Societies of Great Britain and Ireland, it is only comparatively recently—indeed, rather less than a year ago—that the different examining bodies requested the assistance of the medical profession, who thus became aware of the active preparation of a new Pharmacopœia.

Having been Secretary of the Pharmacopœia Committee of the General Medical Council when the present issue was published in 1898, I was invited in that year by the Apothecaries Society to give a short course of lectures on the subject. In these lectures I dealt with what had already been accomplished. Much trouble had been taken to effect improvements, and I endeavoured to set forth concisely the way in which the various changes might affect medical students, medical men, and practical pharmacists.

To-day I have been asked to address this meeting upon the suggestions which are being collected for the next Pharmacopœia, and in this connexion I think some time may be profitably devoted to the *scope* and *object* of the Pharmacopœia. To those actively engaged

upon this work it is evident that there is a widespread lack of appreciation of the scope and object. Perhaps it is more frequently blamed for not being what it is not, than for being what it is. Younger practitioners, who have derived their knowledge of *materia medica* from current text-books, often complain that there is no indication of the uses of the different remedies; this, strictly speaking, is not quite accurate, since there are in the suggestions for doses sundry indications of differences of action with large single doses or with small doses frequently repeated. Still, it is true that the *British Pharmacopœia* is not, and does not pretend to be, the equivalent of a text-book on the actions and uses of drugs, but, on the other hand, it is the source from which the compilers of these books must derive a large part of their information.

Others complain that it does not supply all desirable information upon many practical points connected with prescribing and dispensing. Incompatibility is not stated in the *Pharmacopœia*, and there are very few hints as to methods of compounding or as to the most convenient methods of administration.

On the other hand, it is charged with containing a mass of useless material relating to drugs which are never prescribed, while omitting all notice of many new remedies which are continually employed. Most of these criticisms rest upon an imperfect knowledge of the object of the *Pharmacopœia*, and of the conditions under which it is produced. It is comparatively easy to find fault with the book. It is easy to forget that many drugs whose absence is deplored were not in existence in 1898, and therefore could not possibly be included; while others, which were then praised in some quarters, have already passed into oblivion. The volume is produced "by authority," and, since its authority must be duly respected, it should be generally satisfactory to those for whose guidance it is prepared.

The scope and object of the *Pharmacopœia* are concisely defined in the words of the Act which charges the General Medical Council with the duty of publication.

"By the Medical Act of 1858, section 54, it is enacted¹—'That the General Council shall cause to be published under their direction a Book containing a list of medicines and compounds, and the manner of preparing them, together with the true weights and measures by which they are to be prepared and mixed, and containing such other matter and things relating thereto as the General Council shall think fit, to be called *British Pharmacopœia*; and the General

¹ From Preface, 1867.

Council shall cause to be altered, amended, and republished such Pharmacopœia as often as they shall deem it necessary.'"

Before this date (1858) different Pharmacopœias had been in use in England, Scotland, and Ireland, and when the British Pharmacopœia was to be substituted for these throughout Great Britain and Ireland, the Council had the task of reducing to one standard the processes and descriptions of three different Pharmacopœias and of "reconciling the varying usages in pharmacy and prescriptions of the people of three countries, hitherto in these respects separate and independent." Reference was made to the difficulties encountered in this important work of amalgamation and to the reconciliation of national differences, in some cases at the cost of *mutual concession*.

To quote further from the Preface of the British Pharmacopœia of 1867, to show the spirit actuating the compilers at that date: "The Pharmacopœia having for its object not so much the selection as the definition of substances which the physician prescribes, and which are required to be kept at one safe and uniform standard of strength and composition, some remedies may have been retained in it which have ceased to be in general use, and others introduced, the value of which, although well attested, has not yet been generally recognized." In this will be seen a regretful confession of the necessity of paying due deference to the state of practice—of following rather than leading—of respecting the drugs which are on the wane, while recognizing some which have scarcely yet obtained the foothold which the compilers thought they deserved.

The intention originally was to afford to the members of the medical profession and to those engaged in the preparation of medicines throughout the British Empire one uniform standard and guide, whereby the nature and composition of substances to be used in medicine might be ascertained and determined.

To be a satisfactory "standard and guide" it is necessary to be acquainted with the requirements of those for whom the volume is prepared. The first essential, then, is that the "list of medicines and compounds" should reflect the current ideas of those practising medicine. Opinions regarding the value of drugs are constantly undergoing change and revision, and it is accordingly important that this should be recognized and the ground cleared of useless lumber. No mere respect for antiquity should justify the retention of the obsolete. Again, new discoveries are continually being made, new methods of treatment suggested, and new drugs, especially synthetic compounds, are being

produced with rather bewildering frequency. Those who have tried some of these substances feel that they should be included, and undoubtedly room should be found for drugs which have gained general credence.

Are we all agreed upon which drugs are useless to retain and which should of necessity be admitted? In any meeting of medical men it is little short of amazing to find what a diversity of opinion there is upon such a simple straightforward question. And further it must be remembered that the British Pharmacopœia, although primarily concerned only with medical men and pharmacists, has in course of time had to widen its scope to attempt to satisfy growth in other directions. If "mutual concessions" were needed in the initial stages, they are found to be far more necessary now. In 1898 many of us thought of the burden imposed on the medical student, and steps were taken to facilitate the use of the Pharmacopœia, by altering the strengths of numerous preparations so as to enable the dosage to be arranged upon an intelligible basis. It was hoped that in this way freedom of prescribing might be encouraged, and that the tendency to use proprietary preparations, according to the directions on the bottle, might be checked. (I do not wish this remark to be misunderstood. I hold no brief against proprietary preparations, but I consider it essential that the practitioner should know what he is using, and that the composition of such preparations should be constant.)

In recognizing the medical student as a factor a departure has been made from the comparative simplicity of the original plan. The original structure might almost be likened to a party wall in which the neighbours are equally interested; we might imagine it to be constructed for the safety and comfort of neighbours, even when they might not be on speaking terms. The Medical Council might have been regarded as the landlord who constructed the party wall, after duly consulting the requirements of the tenants on either side as to the materials to be employed. Obviously the members of the medical profession and those engaged in the preparation of medicines would be the interested neighbours. If the party wall merely had to serve the requirements of two individuals, or even of two groups of near neighbours, the construction and the choice of materials might be relatively easy. It must be remembered, however, that the British Pharmacopœia establishes a standard and guide, not only in Great Britain and Ireland, but also in various parts of the British Empire. Since 1898 an Indian and Colonial Addendum has been prepared, and it has been found necessary not only to include many drugs with local reputations, but also to make many modifications

in the directions for compounding pharmacopœial preparations. Some of these modifications were required on account of climatic conditions, while others were necessitated by the limitations imposed by caste.

Further, it is, to say the least, convenient and important that, since most foreign countries have pharmacopœias of their own, there should be no wide divergencies between the standards adopted by the different countries. This is a matter which has in recent years attracted considerable attention, since it is obviously dangerous for the same or similar names to be employed in different countries for galenical preparations of different degrees of activity and of dosage. It is conceivable that prescriptions written in one country may be dispensed in another, and without considerable forethought on the part of the dispenser an overdose may easily be given.

It is perhaps as yet scarcely generally known that an International Agreement has been signed to secure greater uniformity in preparations containing powerful active ingredients. In the construction of the British Pharmacopœia this agreement must be kept in mind, even though it may alter the composition of many preparations to which we have long been accustomed. Happily the changes entailed in the British Pharmacopœia are comparatively slight, and are not likely to disturb practical work to any great extent. Owing to the efforts of the President of the General Medical Council, who, as Chairman of the Pharmacopœia Committee, attended the International Convention, and owing also to the care which was taken in the preparation of the current British Pharmacopœia, the general trend of the alterations recommended was in the direction of adopting the British standard for general use. While some changes, then, must be made universally to produce greater uniformity, it is satisfactory to feel that our own official preparations will be far less affected than those of many European States.

To repeat what I said at the beginning of this address, the ten years that have elapsed since the publication of the British Pharmacopœia have really been ten years of preparation for its successor. Within six months it was felt that some of the criticisms called forth by its appearance should be investigated and reported on by experts, and that arrangements should be made for carrying out such investigations, and also for accumulating information for the publication of the next Pharmacopœia. The Pharmaceutical Societies of Great Britain and of Ireland were invited to appoint representatives to confer with members of the Pharmacopœia Committee, and the result was the formation of an advisory conference in pharmacy and the commencement of a large number of

important researches. Another innovation was the authorization of the publication of reports received from experts in pharmacy and pharmaceutical chemistry upon matters deemed worthy of investigation. By this publication it was hoped that all reports might be criticized, and that any necessary readjustments might be made long before the time when the results were to be included in a new Pharmacopœia.

It is not necessary here to refer in detail to all the subjects investigated, but I may perhaps mention two important groups which were dealt with in the Pharmacy Research Laboratory under the immediate supervision of Professor H. G. Greenish: (1) The determination of the accuracy or otherwise of the Solubilities mentioned in the British Pharmacopœia; (2) the percentage ash of various crude drugs and their powders, regarded as a test for their purity. Meanwhile a new series of researches were necessitated in an unexpected direction. The Arsenical Commission, in the course of its inquiries, had drawn attention to the need of revising the tests for arsenic as an impurity in certain drugs, and Professor Dunstan, at the request of the Council, dealt with this subject in a report which suggested the tests to be employed and the limits of impurity which should not be exceeded.

In 1905 it was felt that more rapid advances might be made by organizing in a more systematic manner the various inquiries and researches that were still necessary for the effective revision of the Pharmacopœia, and it was determined to appoint Committees of Reference to advise upon Chemistry, Botany, Pharmacology, and Pharmacy. A Committee of Reference in Pharmacy was appointed, consisting of expert pharmacists, nominated by the Pharmaceutical Society of Great Britain and by the Pharmaceutical Society of Ireland. At the first meeting of the Committee of Reference in Pharmacy Mr. Walter Hills was chosen as Chairman and Professor Greenish was appointed Secretary. To this Committee of Reference in Pharmacy the International Agreement respecting the Unification of the Pharmacopœial Formulas for Potent Drugs, signed at Brussels in November, 1906, was referred for consideration, and a valuable report has been received dealing with the changes in the Pharmacopœia which must be made in order to make it conform to the provisions of the agreement.

The foregoing statements indicate briefly the lines of work which have been in progress. It will be seen that they deal mainly with questions of pharmacy which entail expenditure of time in research. They are intended to clear the ground, and, unhappily, it must be obvious that much of the labour may be almost wasted, since it has been impossible

as yet to indicate which substances are likely to be deleted. It is necessary to work upon groups, assuming that every substance in the group may be retained.

To revert now to the important question of the construction. As you know, this is entrusted to the General Medical Council, which has appointed a Committee of its members to supervise the whole work. The General Medical Council is composed mainly of representatives of the different examining bodies in Great Britain and Ireland; all of these bodies have, therefore, a direct advisory duty in the construction of the Pharmacopœia, and they are all requested to furnish suggestions for omissions, for additions, and for alterations in the volume. These suggestions are carefully tabulated, and will, in due course, be closely considered. I must not disclose any details at the present time, when the whole question is still *sub judice*, but I think you will be somewhat surprised to hear that a very considerable number of articles in the current Pharmacopœia have been black-balled by one or more of the authorities from whom replies have been received. This curious result is apparently due to the different points of view held by various bodies regarding the scope and object of the British Pharmacopœia.

It is urged by many advisers that duplication of preparations should be avoided, and, as an abstract principle, this seems reasonable and attractive; but when we attempt to act upon this suggestion difficulties at once arise. For example, there may be two or more liquid preparations—solutions—of an alkaloid; these may be made from different salts of the alkaloid, and would therefore come under the general principle above mentioned. Which of them should be retained? The practitioner would probably answer: "The one most commonly used." This may not, however, be altogether satisfactory. The others may be employed with sufficient frequency to require definition, or there may be good pharmaceutical grounds for their retention. They may be present in some galenic preparations, when the one "most commonly used" would not be compatible with other ingredients. As an abstract principle it is admirable to avoid duplication, but as a practical matter it may lead to great confusion.

To give another illustration of the difficulties encountered in the construction of the work, it would seem desirable to eliminate all substances devoid of action which can be determined by physiological experiment or by therapeutic observation. It is well known to experimental pharmacologists that several drugs do not possess the powers with which they are commonly credited. It would seem that they should be removed

from the Pharmacopœia. Students would hail this proceeding with delight. When, however, an attempt is made to ascertain from medical men and from pharmacists whether these substances are in demand at the present time, we may be surprised to find that they are still prescribed with a degree of frequency which necessitates their retention.

The British Pharmacopœia is not empowered to instruct the medical profession what drugs should be employed; it can only note that certain drugs are commonly used, and then endeavour to establish a standard of purity and uniformity of composition. This standard should be a workable standard, and hitherto the intention has been to introduce tests which could be performed by any competent practical pharmacist who desired to ascertain the degree of purity of the drugs supplied by wholesale firms.

Amongst the difficulties with which the Committee will have to contend, one of the greatest, in my opinion, is to determine whether the various sera and vaccines are to be introduced. Some of them are doubtless largely used by most medical men, and therefore it may well be argued that they should be included. If included, however, it is necessary not only to give a description of the manner of preparing them, but also of the tests for their activity and purity. This would involve more training in research work than falls to the lot of most pharmacists, and it would, I think, be necessary to follow the principle adopted in the German pharmacopœia, and to define the activity in terms of a special official laboratory. This would mean that a special State Laboratory must be equipped for this purpose, and that an official guarantee would in future be substituted for personal responsibility.

Another question which will probably demand most careful consideration really almost originated within the Therapeutical Society: I mean the physiological standardization of drugs for which no satisfactory chemical mode of standardization is at present known. I need hardly remind you of the admirable paper on this subject read by my colleague, Professor Dixon, or of the resolution passed by the Society recommending the introduction of physiological standardization. This test, like that for the antitoxins, is not capable of being checked by the average pharmacist, and would also require the establishment of a State Laboratory. If such official laboratories were already in existence the matter would be perfectly easy, but it appears to me, speaking purely in a personal capacity, that the duties imposed upon the General Council under the Medical Act of 1858 do not provide for the creation or the recommendation of such innovations.

I have referred already to the assistance afforded by the replies received from the different examining bodies, and to my inability here to enter into any details. It is satisfactory to note that in several instances the suggestion is made that as little as possible should be altered, though some few new remedies might be added. On the other hand, as I have said, some of the examining bodies and numerous individual critics have tendered advice which would alter nearly every monograph in the Pharmacopœia. The pity is that the advice tendered does not agree in all cases, so that the work of sifting and selecting must be great.

Even in dealing with the formation of the different Committees of Reference the Pharmacopœia Committee recognized that there might be some lack of unanimity in the recommendations received, and put the following upon record:—

"The Committee, who by the Standing Orders have charge on behalf of the Council of all matters relating to the preparation and publication of the Pharmacopœia, think it desirable to state that they reserve their full freedom of action as to the adoption or modification of any proposal that may be made to them by any of their Committees of Reference."¹

Æsop long since indicated the probable dangers involved in attempting to act upon every suggestion—I forget the moral of the fable; but if applied to the Pharmacopœia, I think it should be that while it will certainly be impossible to satisfy everyone, much may be done by mutual concessions and goodwill.

DISCUSSION.

The PRESIDENT (Dr. Burton Brown) expressed the thanks of the Section to Professor Tirard for his interesting and most important contribution, and remarked that formerly the Pharmacopœia was in Latin only, and from such he himself was taught in his student days. He exhibited a copy of the 1689 edition, which also was entirely in Latin.

Dr. C. O. HAWTHORNE thought the Section was to be congratulated on having secured such an authoritative introduction to the discussion. Dr. Tirard had advanced a number of propositions in regard to the function of the Pharmacopœia with which he (Dr. Hawthorne) found himself in entire sympathy. Yet in some parts of the paper it was possible to detect a note of regret that the Pharmacopœia was not constructed on a very different model. There were some authorities who desired that the Pharmacopœia should contain only those remedies which had passed the lists of the pharmacological laboratory or had gained the approval of experts; it had also been proposed

¹ Report of the Pharmacopœia Committee, May 25, 1905.

that the book should supply suggestions for the combination and use of remedies so as to make it "useful" to medical practitioners. Dr. Tirard had not expressed any approval of these proposals, yet in his remarks on the therapeutic value of the official doses, and on one or two other points, he appeared to have a certain measure of sympathy for them. Now in his (Dr. Hawthorne's) opinion these propositions involved a misconception of the true purpose of the *Pharmacopœia*, and if they were put into practice they would, he believed, exercise a mischievous effect on medical practice. The *Pharmacopœia* was a volume issued by the authority of an Act of Parliament, and its sanction must therefore be found in the promotion of some public interest. It would be difficult to believe that the public welfare needed the publication of a volume of therapeutics either by Parliament directly or through the agency of the General Medical Council. Neither the one nor the other of these constituted authorities had any capacity or title to proclaim which drugs had and which had not therapeutic value. Any attempt on their part to do so would lead to the erection of an orthodox standard of practice and to the invasion of the liberty and responsibility of the individual practitioner; and this most certainly would not be in the public interest. Further, there was no need for an official volume on therapeutics, seeing that through the medical press any individual who had experiences or suggestions to announce had full liberty to do so; and truth was promoted, as most of them believed, rather by free discussion than by authoritative proclamations. Thus it was evident that the issue of a *Pharmacopœia* by the authority of Parliament could not find its explanation in the public or professional need for instruction in therapeutics. On the other hand, there was one function which the official volume fulfilled and which it could not fulfil unless it had Parliamentary sanction. That function was the definition, both quantitative and qualitative, of the names of drugs as written in physicians' prescriptions. Manifestly it was to the public interest that terms so used should carry one and the same meaning to every dispenser. Without this the writing and dispensing of prescriptions would involve confusion and even danger. Hence to protect the public Parliament authorized the issue of a *Pharmacopœia* in which drugs and their preparations were defined, and compelled both physicians and pharmacists to understand these terms in the pharmacopœial sense and in that sense only. No authority short of Parliament could compel the universal acceptance of this series of definitions. The public welfare necessitated such acceptance, and therefore Parliament instructed the General Medical Council to prepare and issue the *British Pharmacopœia*. With such a view of the true function of the *Pharmacopœia* it became obvious that admission of any drug to its pages did not imply any guarantee of therapeutic efficacy. Such admission was necessary when a drug was more or less generally prescribed, and it was necessary, not for the instruction of medical students or practitioners, but for the protection of patients for whom the drug might be ordered. Thus, in its proper sphere, the *Pharmacopœia* was a useful, or even necessary apparatus to secure the public interest. To treat it as a text-book for students or as a manual of pharmacological or therapeutic importance was to misapprehend its meaning. In its present subordinate position it

was a useful servant, and the medical profession, if well advised, would never allow it to obtain the rôle of a master.

Mr. WIPPELL GADD said he had been very glad to hear the generous tribute which the author had paid to the Pharmaceutical Society and its work in connexion with the Pharmacopœia. The whole mischief of past Pharmacopœias had been that those responsible for their production had not taken to heart the words of Sir Frederick Pollock in another connexion, that those who made the shoe did not feel it pinch, and those who felt the pinch did not know how shoes were made. Medical men knew what they wanted to prescribe, and pharmacists knew how to put together such medicines as were prescribed. If they did not speak a common language there was sure to be disaster. He was rather sorry to hear the simile about the party wall. He (Mr. Gadd) took it that nothing should be set up between two bodies of men who were working for the health of the people. He had been pleased to hear the remark about the physiological testing of certain drugs in Government laboratories; it had long seemed to him that that was the only solution of the problem. Those who produced such substances were often grieved to think how difficult it was to guarantee to the medical man that they always supplied something uniform, and that was because their chemical tests failed. He referred to such things as *strophanthus*, *digitalis*, and *ergot*, which could only be tested by physiological experiments, and chemists were not licensed vivisectioners. It was desirable that there should be some central laboratory to which such things could be sent. Of course one would like to hear some of those state secrets which the author had alluded to, and he supposed the Committee had considered the effect of the Pharmacopœia as a standard for domestic medicines; it was taken by his Majesty's judges as being a standard for such purposes, or at least as very strong evidence. In the case of *Dickens v. Randerson* it had been laid down by Justices Bruce and Phillimore that when a substance was asked for by its pharmacopœial name it must be supplied of pharmacopœial strength. The article in question was mercurial ointment, and he thought it would be agreed that it was a danger to his Majesty's subjects that that ointment should be promiscuously supplied to the public of the official strength. He wished to know whether the Committee had taken into consideration that secondary use to which the Pharmacopœia might be put.

Professor TIRARD, in reply, said he feared that Dr. Hawthorne had scarcely given him credit for trying to be impersonal in his remarks. As Chairman of the Therapeutic Committee he had endeavoured to be impersonal all through, a statement which the President, as a member of that Committee, would bear out. He carefully refrained, owing to his official position, from attempting to do more than advise, with regard to some of the difficulties which the list of suggested omissions and additions might entail on the Pharmacopœia Committee. Although he was partly responsible for this list, he had tried to show that the difficulties might be very great, and indeed insurmountable. He hoped Dr. Hawthorne was not correct in saying he (Professor Tirard) had expressed a note of regret that the Pharmacopœia was not a text-book of pharmacology and therapeutics. He had tried to show by his quotations that

the authority and scope of the Pharmacopœia were laid down by Act of Parliament; that the Council was empowered merely to prepare a book containing a list of medicines and compounds and the manner of preparing them. Nothing was said about action, pharmacological or therapeutical. The Committee endeavoured to ascertain whether drugs were used; if they were used, whether it was a matter for regret or not, they would have to go in. It might be asked, Why should the Committee feel regret? He thought the answer was to be found in the question raised by Mr. Gadd—the fact that the British Pharmacopœia was a standard of medicines, and there was a growing tendency to employ it as such in the Law Courts. Dr. Hawthorne had asked under what authority it was issued. He (Professor Tirard) gave the authority, namely, the Medical Act of 1858, which gave the authority to the Medical Council, and insisted on that Council performing its functions. That authority merely gave the Committee power to produce that book of medicines and compounds and their composition and their weights and measures. With regard to doses, on page 18 of the Preface it was said: "The doses mentioned in the Pharmacopœia are intended to represent the average range in ordinary cases for adults; they are meant for general guidance, but are not authoritatively enjoined by the Council. The medical practitioner must act on his own responsibility as to the doses of any therapeutic agents which he may administer." In admitting the doses to the Pharmacopœia, they were carefully guarding themselves against exactly the point raised by Dr. Hawthorne as to whether, for example, a dose of quinine was sufficiently large or sufficiently small. The dose of 1 gr. to 10 gr. was intended to be for ordinary cases in adults, and should not be exceeded for them. All would admit that the pharmacopœial dose did not take account of the enormous quantities which might be necessary for malaria; those were not average cases. Those who produced the Pharmacopœia did not attempt to establish an official pharmacology. In his remarks concerning the unnecessary duplication and deletion of drugs which were devoid of action, which could be determined by either experiment or by therapeutic observation, he intended merely to say that that was advice which was given by many bodies, but it was not necessarily advice which could be followed. As he had mentioned, a large part of the advice must, regretfully, not be acted upon, and he thought he had given reasons why. In answer to Mr. Gadd, it was because the Pharmacopœia was so likely to be used in courts of law that it was not desirable it should include substances used in domestic medicines, instead of by practitioners. If a substance was used both by a medical practitioner and in domestic medicine, then if asked for by its pharmacopœial name its composition should always be of the same strength. In that sense great care had to be taken as to what was included, and especially as to what synonyms were mentioned. The intention was to act for the safety of the public, to ensure that they should always receive a substance of definite and uniform strength. In conclusion, he thanked the Section for its patient hearing. The subject was one in which he was keenly interested, but it was one on which he always endeavoured to put his own feelings in the background, to act up to the spirit of the legislation which forced the compilers of the Pharmacopœia to follow rather than to lead.

Therapeutical and Pharmacological Section.

November 3, 1908.

Dr. T. E. BURTON BROWN, C.I.E., President of the Section, in the Chair.

The Treatment of Acute Toxæmias.

By W. E. DIXON, M.D., and HENWOOD W. HARVEY, M.D.

VERY little is known as to how drugs act on diseased organs, and here it should be mentioned that by diseased organs is meant those which are showing some form of inflammation or degeneration. Chronic mitral disease, for example, does not come under this category, since in this condition there is only the scarring of an old disease, and a heart with an incompetent mitral valve behaves towards a drug in precisely the same way as does a normal heart. Thus the action of digitalis in this disease is exactly similar to its action in health. Or, again, prussic acid, cocaine, and atropine have a stimulant action on the brain; in cerebral tumour and in many other forms of cerebral disease, these drugs produce their effect in the ordinary manner. Therefore in all diseases in which there is no degeneration or affection as a whole of the living cells of the tissue upon which drugs act, such drugs exercise, generally speaking, their ordinary physiological actions.

Toxæmia is a somewhat difficult word to define, because there is such a variety of toxæmias not in any way related to one another. It is permissible to speak of toxæmia due to an excessive internal secretion; thus it is generally believed that Graves' disease is the result of a hypersecretion of thyroid juice. We believe that hypersecretion of adrenalin may occur, and we know that such a condition will cause well-marked symptoms. Of these chronic toxæmias we are not proposing to speak, but rather of the acute toxæmias occurring as the result of the absorption

of products of bacterial action, or the result of poisoning from some relatively simple substance such as lead, or the absorption of relatively simple chemical substances of putrefactive origin from the gut. The absorption of lead produces a certain toxic effect, especially upon the muscular system of the body, resulting in hypertrophy of the heart and arteriosclerosis. We might here also refer to nicotine. Nicotine produces a curious toxic effect. We show a chart of the effect of nicotine because it is typical of what probably occurs in a number of other toxæmias caused by absorption from the alimentary canal.

Fig. 1 is the record of the blood-pressure of a boy, aged 17, who was required to smoke a manilla cigar. He had never smoked before, and therefore showed no nicotine immunity. At first the blood-pressure will be seen to rise, but this is soon followed by a great fall, inducing all the well-recognized symptoms of collapse. Fig. 2 is the chart of another boy, who was a year younger and who smoked cigarettes habitually. He also shows the typical rise of blood-pressure as the result of smoking, but it was not succeeded by the great drop as in fig. 1. This is the ordinary effect in the case of a moderate smoker. Fig. 3 shows the blood-pressure of a man who was quite tolerant to tobacco-smoking, so that even double the former amount produced no effect upon his blood-pressure. But it is not with these forms of toxæmia that we wish to deal to-day, but rather with those forms produced through the agency of bacteria. There are many toxic bacterial products which are absorbed from the alimentary canal; some of these are extremely poisonous and have been isolated chemically pure, and some of them exert remarkable powers of raising the blood-pressure. The interest of these substances to us is that they are absorbed with extraordinary rapidity in the presence of alcohol, and to our mind the significance of alcohol as a poison depends largely upon this property of facilitating the absorption of toxic substances.

The toxins, however, to which we desire to draw particular attention to-day are more complicated than these; they are but two: cobra venom and diphtheria toxin. We have chosen these two because we think they are fairly representative of the large group of substances causing acute toxæmia. It has been our object to determine whether there is any difference in the action of a drug on a normal organ and on one which is diseased or which has undergone degeneration as a result of poisoning by one of these toxins. It may be well, in the first place, to mention how the toxins act. It is now generally accepted that both the toxins under consideration produce their effect by combining directly with the

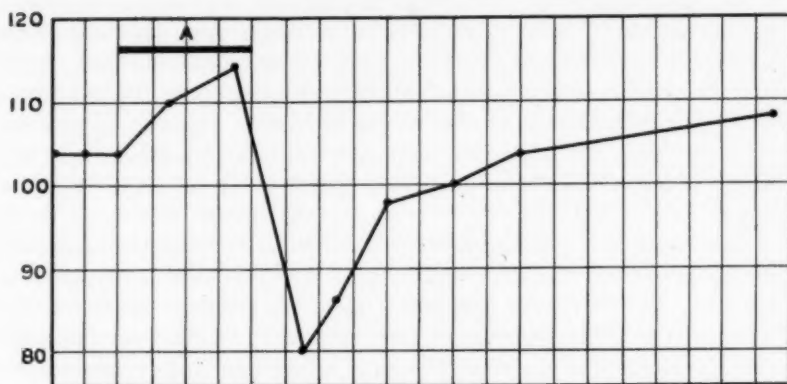


FIG. 1.

Ordinate equals blood-pressure in millimetres of mercury. Abscissa equals five minutes.
A represents the period of smoking.

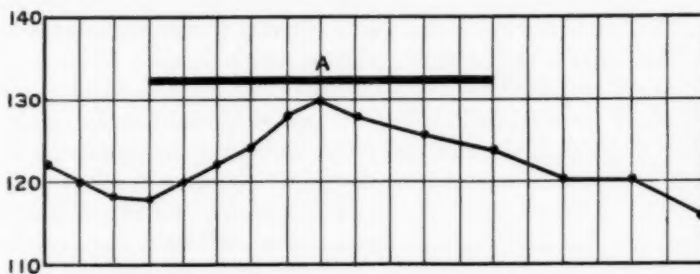


FIG. 2.

Ordinate equals blood-pressure in millimetres of mercury. Abscissa equals five minutes.
A shows the period of smoking.

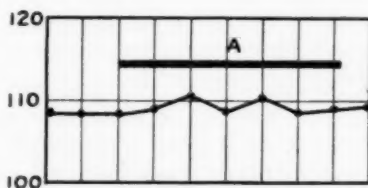


FIG. 3.

Ordinate equals blood-pressure in millimetres of mercury. Abscissa equals five minutes.
A shows the period of smoking (after Lee).

tissues upon which they act. They are believed to combine with those tissues because, if some of either toxin is added to an emulsion of the tissue, the toxin disappears and the mixture becomes non-toxic. Thus, if tetanus toxin is added to an emulsion of nerve-tissue, the mixture is rendered non-poisonous to animals on injection. Ehrlich explains this by saying that the toxin has combined with the cytoplasm, and as a result of the combination destruction of the toxin as a free substance occurs.

The method of experimenting was as follows: Animals, usually cats, were injected subcutaneously with toxin. The dose was so arranged that the next day they were completely under the influence of the poison. The animals under experiment take no interest in their surroundings; they gradually pass into coma and die. A few hours before they reached the comatose stage, about twenty-four after injection, they were anaesthetized and records of blood-pressure, respiration, &c., were taken upon a moving drum. In the case of cobra toxin the injection need only be made after the animal has been anaesthetized, since the toxin acts rapidly, usually in from half an hour to three hours, but in the case of diphtheria toxin the injection must be made at least twenty-four hours before the records are taken.

Cobra toxin, as is well known, destroys certain centres in the brain, particularly the respiratory centre; associated with these effects are certain blood-changes, with which we are not immediately concerned, and finally there is an action on the heart resulting in cessation of the beat. When the respiratory centre first begins to be affected, or shows a tendency to fail, then no drug which we have examined so far possesses the slightest effect on respiration; hydrocyanic acid, caffeine, strychnine, and cocaine, all of them powerful respiratory stimulants, are now found to have lost their normal stimulant action. So we may say, if the hypothesis of Ehrlich be accepted, namely, that the toxin has combined with the living molecule, that this combination, at least so far as its response to drugs is concerned, is dead, since drugs cannot affect those cells in any way. In every case in which the dose is sufficient, respiration gradually ceases, and when it has once failed, although the animal can still be kept alive by artificial respiration for an indefinite period, yet the respiration never again takes place naturally. The cells are permanently destroyed. If larger doses of toxin are given, other nerve-cells are paralysed, especially the autonomic nerve-cells, those on the course of the splanchnic fibres, and the vagus cells.

There is one group of drugs which we have convinced ourselves

produces some effect in this condition. If cobra toxin combines essentially with nerve-tissue, it should be possible by injecting nerve tissue, either in combination with the toxin or soon afterwards, to produce some effect on the disease. As a matter of fact we have tried practically all the substances which have been extracted from the brain. We tried cholesterin, and that has some effect, in that, as is well known, it prevents hæmolysis. It produces, however, no effect on the respiratory centre when once the depression of that centre has set in. We tried lecithin and bile-salts, and, lastly, that complicated substance which is not pure—cerebrin. This cerebrin does neutralize the toxin in the same way as antivenom does. If the cerebrin is mixed with the toxin the effect of the latter on the respiratory centre is neutralized. If some slight depression of respiration only is produced, and then large doses of cerebrin are injected, it is often possible to prevent death in the animal under experiment. It is the only substance which produces any definite effect. Therefore, so far as our experiments go, we believe that, when once the respiratory centre is paralysed, no drug or Galenical preparation has the smallest effect on the respiratory centre.

The second group of toxins which we investigated is represented by diphtheria toxin. It is generally supposed that death occurs in one of two ways: either the heart stops or the respiration ceases; but diphtheria toxin causes death frequently in yet another way. The respiratory centre does not give out and the heart does not cease to beat, but the blood-vessels throughout the body become enormously dilated, the splanchnic area especially being gorged with blood. As a result of this there is a great fall of blood-pressure, and respiration fails because there is too little blood passing through the medulla to keep it in a state of activity. Diphtheria toxin first affects the nerve-tissues, so that they cease to respond to drug action. Hydrocyanic acid, cocaine, and caffein produce no effect on the respiratory centre or vasomotor centre in the later stages of toxæmia. The vagus responds little or not at all to electrical stimuli, although the animal may still be breathing naturally; the sympathetic system throughout the body is active, though less so than normal. The nerve-cells in the spinal cord cease to respond to stimulation, and even very large doses of strychnine produce little or no effect, whilst in the last stages enormous doses of this drug may be given without any effect. It is clear that when the cord is under the influence of this toxin, strychnine and allied drugs cease to exert their normal effect.

The nerve-cells on the course of the splanchnic fibres (vasomotor nerves) become paralysed, so that nicotine, which under normal conditions produces a large rise of blood-pressure by stimulating these cells, now ceases to have any effect, whilst adrenalin, which also acts on the nervous mechanism, but more peripherally than nicotine, still produces a great rise of blood-pressure. Lastly, the motor nerve-endings are destroyed by diphtheria toxin.

If an animal is killed with diphtheria toxin it is possible to extirpate the heart and fix it to a perfusing apparatus, where it will continue to beat so long as it is perfused with saline at sufficient oxygen pressure. The heart itself is not dead at this stage, which is the stage when death occurs in the intact animal. As we have pointed out already, it ceases to beat on account of the enormous dilatation of the vessels, so that there is only a very low blood-pressure and, therefore, a deficient coronary circulation.

It should be our object, then, under these conditions to do what is possible to raise blood-pressure. Digitalis, ergot, strychnine, are of little or no value for this purpose. The most satisfactory method of producing a profound effect on the blood-pressure is by the injection of normal saline solution. If into the vein of a cat of $2\frac{1}{2}$ kilos, under the influence of diphtheria toxin and with a blood-pressure little above zero, an injection of 50 c.c. of warm saline solution be made, an immediate beneficial effect is obtained. Blood-pressure rapidly rises, the heart beats improve, and the respirations, which may have become asphyxial in character, once more become regular and satisfactory. Indeed, the whole condition of the animal may show very marked improvement. Furthermore this improvement may continue for some hours. If a little glucose or alcohol is mixed with the saline solution the beneficial effect is increased.

The two points we desire to bring forward are: (1) the beneficial effects of saline injections in this type of toxæmia, and (2) the fact that drugs will not affect cells poisoned by toxins to the same extent as normal cells.

DISCUSSION.

The PRESIDENT (Dr. Burton Brown), in the name of the Section, cordially thanked Professor Dixon and Dr. Harvey for their important contribution. The subject was one which must appeal to all, especially those who had worked in India, where 20,000 persons annually died from the bites of cobras and other animals; and if anything could be found to cure the results it would be a great benefit to humanity.

Dr. SOPER said the authors had shown that the blood-pressure was raised by nicotine, but in that light it seemed difficult to account for the depression following so quickly and the subsequent collapse.

Dr. FAWCETT said it was interesting to see the result produced by saline intravenous injection, although clinically the value of it was well known; but in small children it was an almost impracticable method on account of the mechanical difficulties involved in carrying it out effectively. With regard to drugs, his experience was only clinical, but he considered it dangerous advice to give medical men that in poisoning from diphtheria they might inject without harm practically any dose of strychnia. He had certainly seen children very ill indeed with diphtheria to whom repeated injections of strychnia had been given, and who had exhibited definite twitching as a result. He thought that probably the cats used by Professor Dixon and Dr. Harvey in their experiments were in much the same condition as an old person who was in a late stage of a malignant "cachexia," and in whom perhaps a perforation of stomach or intestine had taken place; at the autopsy the peritoneum was found full of pus, and yet there had been no reaction and no symptoms of peritonitis, because the patient was too ill to exhibit any reaction as the result of the stimulation of the peritoneum. Before accepting such a dogmatic statement as the result of experiment on animals, one would like to have the statement controlled by facts relating to human beings in illness. He agreed that digitalis had very little effect in toxæmic cases, but he did not agree that strychnine was a harmless drug, even where patients were very ill indeed.

Dr. J. GRAY DUNCANSON said it was very comforting to those in general practice to hear that the methods of treatment which they had been adopting in the past were found, on experimental investigation, to be the best. For years he had been in the habit of giving copious injections of normal saline solution in cases of profound toxæmia, both by the rectum and subcutaneously, but it was sometimes difficult to tell the exact value of such treatment. In puerperal eclampsia saline injections given by the rectum were of great value, and their utility was increased by adding chloral hydrate and bromide of potassium. Recently he had a patient with marked alcoholic convulsions; he gave him every couple of hours copious rectal injections of saline; in twelve hours the convulsions ceased and the mental condition improved. And in the malarial affections which one sometimes saw, with hæmoglobinuria, such injections combined with a little quinine were undoubtedly of service, and rapidly replaced the fluids which were lost to such an alarming extent in these cases.

Professor DIXON, in reply, said that nicotine always raised the blood-pressure at first, except in the most immune people, those who were thoroughly habituated to it. Afterwards there was a stage of collapse if the dose was sufficient. The person who was moderately addicted to smoking got a slight rise of blood-pressure—8 mm. to 10 mm. of mercury—after moderate indulgence. He quite admitted the difficulty of injecting saline in children, but one important point was that when the body needed saline it was wonderful to what degree the rectum acted as a sponge. He was unable to account for the twitchings mentioned when small doses of strychnia were given, though he did not deny that they might have been due to the strychnia. It was true that in the animals to which he had given it, it was his object to make them as ill as he could, and therefore their toxæmia was very severe, and he would say that in the most severe toxæmias the strychnine did nothing. When moderately under the influence of toxin, more and more strychnine was required to produce an effect, and then a time came when no quantity had any effect.

The Action of Saline Purgatives.

By ARTHUR F. HERTZ, M.D., F. COOK, and E. G. SCHLESINGER.

OVER fifty years ago Buchheim [2] and H. Wagner [10] independently came to the conclusion that saline purgatives owed their power to the fact that they were only slowly and incompletely absorbed in the stomach and intestines. The water of solution, whether taken with the salt by mouth or attracted by osmosis into the intestine, if the salt was taken with little or no water, was believed to pass to the colon, where it produced fluid stools, which still contained the greater part of the salt.

A different theory was proposed by Aubert [1] about the same time. He injected 5 oz. of magnesium sulphate into a horse's vein and found that its bowels were opened soon afterwards. From this single inconclusive experiment he was led to suggest that saline purgatives act only after absorption, the effect depending on a specific irritation of the intestinal nerves leading to increased peristalsis. Numerous experiments performed by Buchheim, Wagner, Hay, Leubuscher, and others seemed to show, however, that the intravenous injection of saline purgatives did not produce purgation, so that Buchheim and Wagner's theory held the field until a few years ago.

In 1901 Loeb observed that the purgative salts are the same as those which increase the irritability of skeletal muscle and nerve. At his suggestion J. B. MacCallum [6] reinvestigated the subject and came to the same conclusion as Aubert, viz., that the saline purgatives only act after absorption, as he found that they produced their effect more rapidly after intravenous injection or direct application to the peritoneal surface of the intestines than after injection into the lumen of the gut. His experiments led him to believe that they act by directly stimulating both peristalsis and intestinal secretion. The experiments of Moreau, Brunton, and Hay had shown that the fluid which collects in the intestines after the administration of a saline purge is succus entericus and not a mere exudation, thus confirming the idea that it is produced by a stimulation of the secretory nerves and not by any purely physical process such as osmosis.

MacCallum's views have not, however, been generally accepted. Meltzer and Auer [8], and more recently Frankl [3], have directly

denied the accuracy of his observations; they state that intravenous injection of sodium sulphate produces constipation rather than diarrhoea.

Our observations by means of X-rays and auscultation on the passage of food along the alimentary canal of man [5] have shown that the cæcum is normally reached about four hours after a meal. Saline aperients may produce an action of the bowels within half an hour of their administration, and they rarely require as long a period as three hours. Hence it is difficult to understand how they can reach the colon by way of the alimentary canal with sufficient rapidity to act from the intestinal lumen, as they are supposed to do according to the commonly accepted theory of Buchheim and Wagner.

In order to investigate this question three individuals took 2 oz. of bismuth oxychloride in $\frac{1}{2}$ pint of cold water at 8 a.m. one morning. They breakfasted at 8.30 a.m. The cæcal sounds and the shadow of the cæcum were first observed at the normal time—about four hours after the meal. Hence the cold water, though taken half an hour before breakfast, did not reach the cæcum very rapidly, although it probably passed out of the stomach without delay. Probably it was completely absorbed from the small intestine, the bismuth being subsequently carried on by the remnants of the breakfast.

A few days later a Seidlitz powder was taken by the same individuals with 2 oz. of bismuth oxychloride and half a pint of water at 8 a.m., half an hour before breakfast. Once more the cæcal shadow did not appear before the normal time, although some cæcal sounds could be heard at a slightly earlier period. The bowels had been opened normally before breakfast, and a fluid motion, the result of the saline purgative, was produced at 9.15, 9.40, and 9.45 respectively in the three individuals—that is to say, about three hours before the first trace of bismuth reached the cæcum and some time before the first cæcal sounds were heard. It might be suggested that the soluble saline purgative traverses the intestines more rapidly than the heavy and insoluble bismuth oxychloride, in which case the first appearance of the cæcal shadow would give no accurate indication as to the time of arrival of the purgative salt in the cæcum. We have, however, proved by observations on a patient with a fistula of the ileum, situated within a foot of the cæcum, that the bismuth salt travels quite as rapidly as the purgative salt. Hence it appears that some of the purgative salt must have been absorbed from the stomach or small intestine into the blood, from which it acted directly on the neuro-muscular mechanism of the colon, producing increased motor and secretory activity in the way described by

MacCallum. The increased activity is apparently confined to the colon, though perhaps the slightly earlier occurrence of cæcal sounds when the salt was taken was due to the production of a secretion in the small intestine, which reached the cæcum in advance of the bismuth and the part of the aperient salt which was still unabsorbed.

We have further proved the correctness of Aubert's and MacCallum's theory by a completely different method, in which the fæces and urine were analysed after magnesium sulphate had been given, and the results were compared with control analyses made on the previous day. The soluble sulphates were extracted and weighed as barium sulphate, and the equivalent amount of magnesium sulphate in the case of the fæces was calculated. It was found that the watery stool, passed one or two hours after a drachm of magnesium sulphate had been taken in $\frac{1}{2}$ pint of water, contained only a few grains more of the salt than the normal solid stool which had been passed earlier in the morning, immediately before the salt had been taken. The largest quantity of water which the salt present in the fæces could have held in the lumen of the gut so as to bring the osmotic pressure down to the level of that of the body fluids, was less than one-third of the amount of water actually present in the stool. It is thus clear that the excess of water present in the stool must have been actively secreted, as it could not have been attracted into the lumen of the gut by physical means alone. No more fæces were excreted until the next morning, when a normal solid stool was passed. This was found to contain a distinctly larger quantity of magnesium sulphate than the more abundant watery stool of the previous day. As the stool was quite solid, the presence of a purgative salt in the lumen of the lower part of the large intestine is clearly insufficient to produce a watery stool, as it should do if its purgative action depended on osmotic attraction. As the magnesium sulphate did not act from the lumen of the gut, it must have acted from the blood. The slight increase in the quantity of magnesium sulphate present in the watery stool cannot, as the bismuth observations show, have been due to the direct passage of the salt from the stomach along the alimentary canal to the rectum. It was doubtless partly due to the increased quantity of succus entericus secreted, as the latter normally contains magnesium salts and sulphates. But the greater part of the excess was probably a result of the excretion into the lower end of the colon of some of the magnesium sulphate absorbed from the upper part of the small intestine, as it is well known that more of the salt is excreted by the mucous membrane of the large intestine than by the kidneys, when it is injected into the blood.

A comparison between analyses of the urine passed on the day on which the magnesium sulphate was taken and that passed on the previous day showed that there was already an increase in the percentage and still more in the total quantity of the total sulphates present in the urine in the four hours following the administration of the salt. Although only a small proportion of the magnesium sulphate present in the blood is excreted in the urine, these observations show that an increased quantity must already at this period have been circulating in the blood.

If the physical theory of the action of saline purgatives were correct, it would be difficult to understand how in some individuals they invariably fail to act. Observations made by us on a healthy man, on whom 1 dr. of magnesium sulphate had no effect, showed that a large proportion of the salt was present in a solid stool passed fourteen hours after it was taken. According to the physical theory it is difficult to understand why excess of water should not have been present in the stool. It is well known, however, that considerable variations occur among different individuals in their power of absorbing inorganic salts. In such cases as this there seems to be a deficient absorption of the salt, so that no aperient action is produced. It passed through the intestines with the food, and some of it appeared in the first stool passed more than twelve hours after its administration, twelve hours being approximately the shortest period which our X-ray observations have shown is required for the normal passage through the intestines. In other individuals the power of absorption may be unimpaired, but the neuromuscular mechanism of the intestine may be less responsive to chemical stimulation than is usually the case.

In our researches on the normal movements of the alimentary canal, we showed that in defæcation the whole colon takes an active part and that all the intestinal contents below the splenic flexure are excreted. In spite of the peristaltic activity of the cæcum, ascending colon, and transverse colon, these parts are not completely evacuated (fig. 1), although a certain proportion of their contents is propelled further along the intestine. When, however, a dose of a saline purgative, sufficient to produce a single copious and semi-liquid stool, is given, the whole of the large intestine from the cæcum to the rectum may be completely emptied. This was well shown in an individual who had taken 2 oz. of bismuth oxychloride with bread and milk at midnight, so that at 9 o'clock the following morning the shadows of the cæcum, ascending colon, transverse colon, and descending colon were distinctly visible with the fluorescent screen (fig. 2). A drachm of magnesium sulphate in

$\frac{1}{2}$ pint of water was then drunk, and soon afterwards breakfast was taken. The breakfast was found as usual to have produced a slight advance in the most forward part of the shadow, but there was otherwise no change in it. Half an hour later the aperient acted, a copious semi-fluid stool being passed. Every trace of a shadow had now disappeared, the cæcum being as completely emptied as the pelvic colon. Observations made in cases of constipation have also shown that saline purgatives produce little or no acceleration in the passage of the chyme along the small intestine, the colon being the part of the bowel upon which the salt present in the blood-stream acts most strongly.

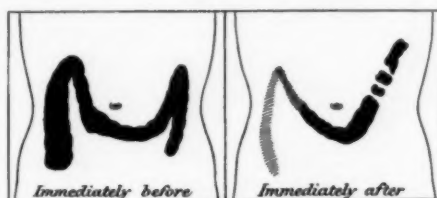


FIG. 1.

Normal defæcation.

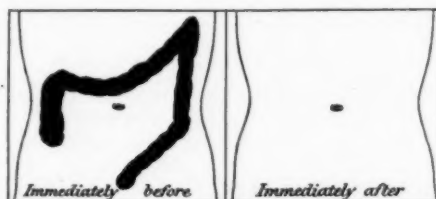


FIG. 2.

Defæcation following administration of 1 dr. of magnesium sulphate.

Thus saline aperients are particularly valuable when it is desired to produce a complete evacuation of the colon without interfering with digestion in the small intestine. They probably have the advantage in such cases over many vegetable purgatives, such as cascara sagrada and castor oil, in producing no acceleration in the passage of the chyme through the small intestine, which would lead to diminished digestion by the pancreatic and intestinal juices. For X-ray observations made in

ordinary cases of constipation, in which the colon is alone involved, and in one due to lead poisoning, in which delay occurs in the small as well as the large intestine, showed that cascara produces increased activity of all parts of the intestines [4], and the same fact was recently observed in the case of castor oil by Magnus [7] in his experiments on cats. On the other hand, he found that senna acted on the large intestine only.

It is still necessary to show how the revived theory of Aubert and MacCallum of the action of saline purgatives can explain various points in connexion with the ordinary methods used in their administration. They are most active when given dissolved in a considerable volume of water, because, as the experiments of Otto [9] have shown, solutions of salts are retained in the stomach until they become isotonic with the body fluids by dilution with the secretion of the gastric mucous membrane. Thus the nearer the solution of the salt is to being isotonic with the blood-plasma, the more rapidly it will pass into the

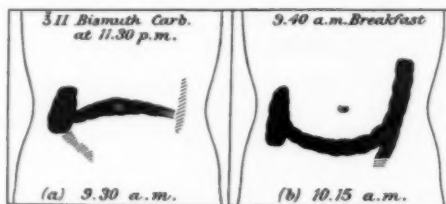


FIG. 3.

Effect of breakfast: (i) emptying of small intestine, (ii) advance in colon, (iii) lower position of transverse colon

small intestine, from which its absorption occurs. It is best given on an empty stomach, as it then passes directly into the small intestine; on the other hand, if food is present in the stomach the salt passes slowly out with the food, so that it may be excreted almost as rapidly as it is absorbed; the result is that no purgative action is produced, but disturbances in gastric digestion often occur, owing to the abnormality in the secretion of gastric juice and the inhibition of ferment action to which it might give rise. Lastly, a saline aperient is best taken a short time before breakfast, because then the specific action of the salt is augmented by the normal stimulation of peristalsis, which, as our X-ray observations have proved, occurs when food is taken into an empty stomach (fig. 3). The importance of this factor is shown by the

observation that in one individual a drachm of magnesium sulphate taken before breakfast produced an evacuation in thirty-five minutes, whereas the same dose given another morning, when no breakfast was taken, required seventy minutes to produce its action.

We are indebted to Mr. H. Marshall for valuable assistance in several of the experiments. We intend to publish full details of the analyses at a later date.

TYPICAL ANALYSIS BY F. COOK.

(Sodium sulphate, 1 dr., taken at 9.40 a.m. on second day.)

	Percentage water	Total SO ₄	Percentage SO ₄
First day, normal faeces ...	80.9 ...	0.037 gm. ...	0.045
Second day, normal faeces (10.15) ...	80.0 ...	0.016 „ ...	0.032
„ watery faeces (11.25) ...	91.1 ...	0.091 „ ...	0.041
Third day, normal faeces ...	77.3 ...	0.270 „ ...	0.230

Urine secreted between 8 a.m. and 4 p.m. on second day contained 0.624 gm. more SO₄ (= 36 gr. crystallized sodium sulphate) than on first day.

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DISCUSSION.

The PRESIDENT (Dr. Burton Brown) thanked the reader of the paper and his coadjutors for their interesting communication. Medical men had long known the proper time to give purgatives, and the present paper showed they were right.

Professor CUSHNY said it seemed that the progress of pharmacology was somewhat in a spiral curve. Pharmacologists had been content for many years with the view that the saline cathartics acted in a physico-chemical way; and, in spite of the recent objections to that view, pharmacologists had not been

convinced by the work of MacCallum. Most of them believed at present that magnesium sulphate and the other sulphates acted simply by delaying absorption. The evidence given by Dr. Hertz was the most plausible which he had heard, and he was scarcely prepared to criticize it without looking at the figures more closely. The objection which struck experimentalists in the matter was, that one could give a dog a saline cathartic by the mouth and defecation followed within a certain time without the slightest question of the effect. If one gave twice as much of the saline cathartic intravenously, there was no movement to speak of; movement only occurred when the bowel was exposed to the air. In the course of work on another subject he injected large quantities of sodium sulphate intravenously in animals, and unless the bowel was exposed there was no movement whatever. The experimentalist could not quite accept the view that those purgatives acted by direct effect on the muscle. How Dr. Hertz's results were otherwise to be explained he would hesitate to say, but in some work done by Wallace, in his (Professor Cushny's) laboratory at one time, the saline cathartic passed as far as the cæcum of the dog within half an hour, *i.e.*, 65 per cent. of what he gave reached the cæcum in that time. One could quite conceive that that was sufficient to induce purgation.

Dr. H. C. CAMERON asked what precautions were taken to ensure that the subjects of the experiments were not influenced by their own emotions. Did they know the importance of what was going on? It was difficult, perhaps, to attain impartiality in such a matter, and in these subjects the bowels might have been opened earlier than in the case of an unintelligent animal in the laboratory. The more crucial the experiment, the greater the force of the suggestion.

Professor DIXON said that seven or eight years ago, when he was working at the subject of hypodermic purgatives, his attention was drawn by clinicians to the fact that they had in their search for hypodermic purgatives used magnesium sulphate, from $\frac{1}{2}$ gr. to 2 gr. or even 3 gr. He (Dr. Dixon) was trying those by injection into animals, into patients, and into himself. They had no purgative effect on man, though undoubtedly they increased peristalsis in animals. It was necessary to be very careful about giving more of the sulphate, because of the extremely toxic effect upon the heart. What struck him about the paper was that if the purgative effect was due to the absorption of the drug, and if that absorption was so small, as indeed it must be or otherwise the patient would be poisoned, why should the dosage so materially alter the effect? For example, with sulphur the action was the same with a small as with a large dose, because only a small amount of the sulphur was converted into sulphide, and this substance caused the purgation. But the effect of the magnesium sulphate seemed to be in direct relationship to the quantity administered. Again, why should there be different effects with different degrees of concentration of the magnesium sulphate? Clinicians said that in some cases it was most useful to give it concentrated, while in others it was better more dilute. He thought Dr. Hertz's main argument was that the

effect came on rapidly. But that did not seem a priori to be a very serious objection, and he agreed with Professor Cushny that it was easy to understand that watery constituents could pass down the alimentary canal in half an hour; it seemed to him unfair to assume that the salt and water moved at the same rate as the bismuth. The bowel was practically empty, and the magnesium sulphate, by collecting to itself some water, rendered the contents of the gut more bulky, so that when it reached the colon, which was the most sensitive part of the alimentary canal, it responded more readily to a bulky stimulus than did any other part. Though great stress had been laid on MacCallum's work, that authority did not initiate it nor describe any new fact. In his (Professor Dixon's) communication which he previously made, references to other work of the same description were given.

Dr. J. GRAY DUNCANSON asked whether Dr. Hertz's subjects were lying down or moving about between the administration of the purgative and the time when the X-ray investigation was made. Magnesium sulphate had a different action in the human being according to whether he was lying down or standing. If a rapid action were desired the patient should take the saline after he had risen in the morning. Dr. Hertz had said nothing about blood-pressure, possibly because he regarded it as outside the province of his paper; but if he had taken any observations on that question, no doubt it would be very interesting to hear them. Matthew Hay said that experimentally the blood-pressure was raised, whereas clinically one had been inclined in the past to give magnesium sulphate to reduce the blood-pressure.

Dr. OTTO MAY said that some years ago he did some experiments on cats which had a bearing on the subject in question. He opened the abdomen under anaesthetics, emptied a loop of gut, and then divided, by ligatures, the gut into three compartments. Into one of them he injected a solution of magnesium sulphate, 5 c.c., six times as strong as the isotonic solution. The second loop was kept empty, and into the third loop he put 5 c.c. of normal saline, and then sewed up the abdomen and left it a couple of hours. The result was that the loop which had contained 5 c.c. of the strong sulphate was distended with fluid—in one case the 5 c.c. had increased to 30 c.c.—whereas both the other loops were empty. In the 30 c.c. he did not estimate the magnesium sulphate quantitatively, but there was much magnesium sulphate left, as shown by a precipitate with barium chloride, and there was no digestive power in the fluid produced in that way. Those experiments, of course, were not conclusive one way or the other, but they suggested that under some conditions a physical action of the sulphate was probable.

Dr. HERTZ, in reply, said that they did not necessarily conclude that saline purgatives acted from the blood by stimulating peristalsis and secretion—the observations were merely very suggestive of that. But it was impossible to explain the experiments on the assumption that they acted in the way which pharmacologists had always believed, *i.e.*, by purely physical means. The experiments were strictly physiological, and the people who were the subjects

of them were normal. There was nothing so grossly unphysiological as injection into veins; the salt was taken in the same way as it would be by a person who wished to get his bowels open. He could not explain why the salt did not act when it was injected intravenously in animals. But the latter experiment was not without fallacies, because it was a most abnormal condition to get the body fluids suddenly overwhelmed with excess of salts. He did not think the point referred to by Professor Cushny about a purgative reaching the end of the ileum in a dog in half an hour was very important, because a dog's intestine was not of the same length as that of man, and it need not behave in the same way. To make a comparison one should ascertain by means of the X-rays how long food normally takes to reach the cæcum in a dog. Moreover, there was a distinct delay at the ileo-cæcal valve. Their observations on a boy with an intestinal fistula showed that the bismuth reached the end of the ileum, whence it escaped as quickly as the purgative salt. Therefore the time of arrival of the bismuth in the cæcum gave a good idea of that of the purgative salt. They had demonstrated conclusively that the sounds over the cæcum were due to the arrival of fluid there. Those sounds were absent from the cæcum until four hours after a meal. When a saline purgative was given, the sounds were heard in the cæcum only twenty minutes earlier than usual. In answer to Dr. Cameron, the experiments were done on people who knew their significance, but they had been confirmed by observations on constipated patients in the hospital, who had no idea that anything out of the ordinary was being done. If there was any question here of the saline acting by suggestion, one might as well say it always did so, because a man who took a saline purgative did so in the hope and expectation that it would act. Moreover, one of the individuals investigated did not have his bowels opened though he took half an ounce of sodium sulphate. In answer to Professor Dixon, he did not see why small and large doses should have the same effect according to their theory, because the larger dose would occupy a longer time in being absorbed, so that its action would be prolonged. As a matter of practice he did not think one could say that doubling the dose doubled the effect. In answer to Dr. Duncanson, the subjects took the saline before breakfast in the ordinary way, moving about afterwards, just as anybody taking a saline purgative would do. The blood-pressure was not recorded. Dr. Otto May's experiments certainly showed that there could be such a thing as direct attraction of fluid into the intestines by the salt, and he supposed that that occurred in the stomach when salts were given in concentrated solution by the mouth. But Dr. May admitted that his experiments could not be directly applied to the taking of salts by a normal person, because they reached the intestines in a much more diluted condition, a comparatively small dose being distributed along the whole intestine. Moreover, in Dr. May's experiment the fluid produced in the piece of gut had no digestive action, whereas other experiments, such as those of Sir Lauder Brunton, had shown that saline purgatives can produce a secretion of succus entericus, and not merely of water.

Therapeutical and Pharmacological Section.

December 1, 1908.

Dr. T. E. BURTON BROWN, C.I.E., President of the Section, in the Chair.

The Action of Salicylates in Rheumatic Affections.

By RALPH STOCKMAN, M.D.

It is now generally conceded that acute rheumatism is a specific fever. The prominent part played by the joint-affections, owing to the pain and helplessness of which they are the cause, have in the past somewhat obscured the proper conception of it as a general blood-infection with local manifestations tending to implicate specially certain tissues and organs, but the high temperature, the leucocytosis, the anæmia, the arthritis, the endo- and peri-carditis, and the tendency to natural cure stamp it as a typical fever with a close general resemblance to many other diseases of the same class. Its duration varies from a few days to three to six weeks. One attack does not confer immunity, but rather predisposes to others. This suggests continued infection such as is seen often in rheumatoid arthritis and tubercle, and sometimes in gonorrhoeal rheumatism, where, after periods of latency, the infected person suffers from acute exacerbations. It is probable that many cases of rheumatic infection are very slight; there may be only sore throat with a trifling rise of temperature and some malaise, or it may take the form of a so-called "chill" with no typical symptoms whatever and rapidly aborting.

In considering ordinary well-marked cases, such as are sent into hospital with the diagnosis "rheumatic fever," it seems to me that two types may be distinguished clinically. In the first the temperature ranges from 102° F. to 104° F., there is much constitutional disturbance, usually several joints are very swollen and painful, there is abundant sour-smelling acid perspiration, and as the inflammation in one joint subsides others become implicated in turn. These cases are very

susceptible to salicylate treatment, they are of shorter duration, show less tendency to relapse, and are less liable to be followed by sequelæ than the others. In the second type the joints are not so prominently affected, but the fibrous tissues about the chest, the lumbar aponeurosis, the fascia lata, the periarticular tissues, the tendon-sheaths, and the subcutaneous fibrous tissue are tender and painful, sour-smelling sweats are absent, the temperature is not so high, and salicylate treatment is not nearly so effective. These cases linger on, relapses are more apt to occur, and sequelæ, such as chronic fibrositis and perineuritis, are much more common.

In the present indeterminate state of our knowledge regarding the specific organism of rheumatic fever, it is impossible to say whether these are two different diseases, or merely differing clinical types of the same disease. Cases of mild pyæmia, of osteo-myelitis, of gonorrhœal arthritis, and of rheumatoid arthritis are often, in their early stages at least, very difficult to differentiate from cases of acute rheumatism, and as yet we have no absolute diagnostic such as is afforded by the bacillus of tubercle or the agglutination test in enteric fever. We have to rely for a diagnosis entirely on clinical experience. In most cases of acute rheumatism it has been found impossible to cultivate an organism from the blood or joint-fluid. It is true that in a few cases diplococci, streptococci, and staphylococci have been obtained from the blood during life, but bacteriologists are not in agreement as to whether these constitute the primary or merely a secondary infection. Post-mortem, similar organisms are often obtainable from the synovial membranes, the heart valves, and the meninges. Achalmé has isolated an anaerobic bacillus which he regards as the specific cause, while it has even been suggested that the organism is a protozoop. The reason for this last view is interesting, although possibly not of much weight—namely, that hitherto only protozoon diseases, as malaria, syphilis, and sleeping sickness, have shown themselves susceptible to drug treatment, while bacteria, in the living body at least, have practically defied specific drugs.

Many competent observers (Singer, Sahli) still hold the view that acute rheumatism is merely a mild pyæmia, and it is quite possible that many cases diagnosed and treated as rheumatic are really pyæmic; but I think we may safely conclude that there is a special non-pyogenic organism, and that it is the cause of a train of distinctive symptoms which go to make up the clinical picture of acute rheumatism.

From its first introduction into practice it has been almost universally recognised that salicylic acid and compounds from which it

can be formed in the body have a powerful and quite specific action in this disease. Some practitioners go so far as to base their diagnosis of a case on its reaction to salicylic treatment. But cases of acute rheumatism react in very different degrees. In some its action is prompt and effective, like the crisis in pneumonia; in others it may be slightly slower, or very much slower; and in certain cases the temperature and general condition remain far from satisfactory. Relapses may even occur while the patient is taking the drug. If, however, there is absolutely no beneficial effect, I think we may conclude that the disease is not rheumatic. In some cases the unsatisfactory results of salicylic treatment are undoubtedly due to the dose being too small. When it is increased each time, or a substantial addition, such as 40 gr., given once daily, matters often mend at once, and the temperature falls. Apart from this, however, those cases where the fibrous tissues are chiefly affected never do so well as those in which the joints are mainly involved. This is susceptible of more than one explanation. The two conditions may be due to different, although allied, organisms which are not equally influenced by salicylic acid. There is nothing impossible in this, as in typhoid and paratyphoid fevers, and in human and bovine tuberculosis, we have much the same clinical symptoms produced by differing microbes. Another explanation is that organisms lodged in the fibrous tissues are more protected or under more favourable circumstances for continued existence than those in the blood and joints, and hence offer more resistance to the drug. Certain cases of pericarditis yield very readily to salicylates, while in others they have practically no effect, and in my mind there is no doubt that here we are dealing with different organisms. I do not refer to cases of pneumococcal or tubercular pericarditis, but to those which are clinically and customarily reckoned as rheumatic.

Once the microbic origin of acute rheumatism and its complications and the specific action on them of salicylic acid are admitted, the logical outcome is to treat cases early and with large doses in order to kill off the organisms while they are still in the blood and joints, and before they have established themselves in the fibrous tissues, from which they are dislodged with very much more difficulty. I believe that "chills" of rheumatic origin are rapidly cured in this way, while those which happen to be of influenzal or other origin derive no benefit from salicylates. The doses need to be large and frequently repeated, partly because salicylic acid is so rapidly excreted, and partly because it is converted into the inert salicyluric acid. There is no danger in

pushing it as regards the circulation, which it affects very slightly, its main poisonous action being exerted on the nervous system and respiration.

The action of salicylic acid in acute rheumatism is undoubtedly specific. It is of little or no therapeutical value in other febrile diseases, and it exerts no action on the healthy man from which one could infer its action in rheumatic fever. But besides clinical evidence, the chemical evidence points conclusively in the same direction. Phenol ($C_6H_5.OH$) has no action on rheumatism, but benzoic acid ($C_6H_5.COOH$), a direct derivative, has a very marked effect only inferior to salicylic acid ($C_6H_5.COOH.OH$) or ortho-oxybenzoic acid, as it is called chemically. Its isomers meta- and para-oxybenzoic acids are practically inert, although possessed of the same chemical constitution, and only differing in the position occupied in the molecule by the hydroxyl group (OH). No explanation has ever been forthcoming for the cause of this remarkable difference in action. The cresotinic acids ($C_6H_3.COOH.CH_3$) are all active, the carboxyl and hydroxyl groups being in the same relative positions as in salicylic acid. Salicin, saligenin, acetyl-salicylic acid, methyl salicylate, and several other substances are active only because salicylic acid is formed from them in the body, while populin (benzoyl-salicin), methyl-salicylic acid, and dimethyl-salicylic acid, from which no salicylic acid is formed in the body, are inert. Phtalic ($C_6H_4.COOH.COOH$) and toluic ($C_6H_4.COOH.CH_3$) acids are also quite inactive. So far no chemical or pharmacological explanation has been forthcoming of the specific effect of salicylic and benzoic acids in acute rheumatism, while so many closely allied substances are inert.

The Effective Treatment of Acute and Subacute Rheumatism.

By D. B. LEES, M.D.

THE title of this paper is intended to imply that the treatment of acute and subacute rheumatism, as advised in the text-books and as usually carried out in practice, is inadequate and unsatisfactory. It is true that a great advance has been made since the days when it was maintained that cases of this disease did as well upon mint-water or upon lemon-juice as with any powerful drugs. In those days, as some of us can remember, there was no great exaggeration in the story of the

physician who was asked "What was good for rheumatism?" and who replied "Six weeks." At that time it was common for rheumatic patients to remain in hospital for weeks, and the very painful and persistent arthritis, with the profuse acid sweats, formed a clinical picture which is now not seen in hospital wards. Fatal hyperpyrexia, now extremely rare, was then not uncommon. There was, however, even then reason for thinking that treatment by alkalies was of some distinct advantage, in spite of the fact that the alkali employed was generally a salt of potash, and therefore not free from depressing effects, and that it was administered only in small quantities. An investigation of the clinical records of Guy's Hospital, which I made in 1874 as the basis of my thesis for the Cambridge M.B. degree, seemed to me to show clearly that the alkaline treatment certainly gave the best results. Yet these results left much to be desired, and the ineffectiveness of the treatment of acute rheumatism was one of the reproaches of medicine.

It is clearly seen by us all how great an improvement has been effected by the introduction of salicylate of soda. The rapid relief of pain, the fall of temperature, and the shortened duration of the illness are results which follow, we all agree, the administration of this drug. Yet the remedy, it must be confessed, has some side-effects which are annoying and which tend to limit its usefulness. Without much investigation whether these hindrances could be removed by some modification of the method of administration, physicians have been inclined to desert the simple salicylate in favour of some of the organic compounds of salicylic acid provided for us by the ingenuity of German chemists. But the most commonly used of these, acetyl-salicylic acid, the so-called "aspirin," cannot be combined with an alkali, hence we are at once deprived of a useful ally of sodium salicylate; and though free from some of the side-effects of salicylate, it may produce the most dangerous symptoms of salicylate poisoning, as proved by the case which I have elsewhere narrated of the girl who became comatose with typical "air-hunger" after seven 15 gr. doses of aspirin given in two and one-third days.

The use of sodium salicylate is also hindered by the superstition still prevalent among us that it is a depressant to the heart. The origin of this mischievous notion was probably due, in the first place, to impurities in the drug when a large demand suddenly arose through its early reputation as an antirheumatic. But the drug as now supplied to hospitals is of great purity, and careful observation of its effects for many years enables me to say deliberately that, when given to a rheumatic

patient, it does not cause any weakening of the cardiac action. But it must be remembered that the rheumatic toxæmia itself always causes more or less cardiac weakening, and that this may sometimes become specially manifest. If at the time salicylate is being taken, the remedy is almost certain to be blamed for what is in reality one of the effects of the disease. It must be borne in mind, first, that a similar cardiac failure occurs in diphtheria and in influenza, quite apart from the action of any drug, and, secondly, that in *every* case of acute or subacute rheumatism the cardiac muscle suffers more or less. Endocarditis occurs only in a certain number of cases, and pericarditis is still less common, but the existence of a dilatation of the left ventricle can always be proved by careful percussion. Since I brought forward evidence of this fact before the Society in 1898 I have never seen a case of acute or subacute rheumatism, in a child or in an adult, in which dilatation of the left ventricle did not exist. It appears to be invariably present in this disease. For a recent confirmation of this fact I may refer to the instructive article on rheumatic myocarditis contributed to the last number of the *Quarterly Journal of Medicine* (vol. ii, No. 5) by my friend and former house-physician, Dr. Carey Coombs, of Bristol.

The idea that sodium salicylate was a cardiac depressant, conjoined with the frequent difficulties in its administration, have caused physicians to give it only in small doses, and to discontinue its use as soon as or soon after the painful arthritis had subsided and the temperature fallen. It is a common experience that with this method relapses of rheumatic symptoms, in one form or another, have been frequent. It is also a common experience that often the temperature does not fall permanently to the normal, but that the evening temperature persistently rises to 99° F. or 100° F., sometimes for a considerable period, especially in children; this should never be disregarded, for it means that the rheumatism is still active. It is therefore clear that the present treatment of rheumatism is inadequate, for though it controls it does not completely arrest the disease.

But it is not merely inadequate; it is also unsatisfactory, for it aims almost entirely at the cure of the painful arthritis, and pays little or no heed to the heart. It despairs of any real influence on the condition of this organ; it has no hope of diminishing a rheumatic endocarditis, and it usually ignores altogether the affection of the cardiac muscle. This is largely due to the fact that the earlier descriptions of acute rheumatism were the result of observations of the disease not in

a children's hospital, but in hospitals for adults. Hence the present absurd arrangement of the text-books, in which a description of its characteristics in adults is followed by an article describing what the writers are pleased to call its "peculiarities in childhood." Yet those who have worked simultaneously both in a hospital for children and in one for adults know well that it is in children that the disease is most frequent and most virulent; that in a considerable number of children the first attack is fatal, and that it fatally cripples the heart in a large number of the cases which survive. If every medical student of the past, and especially the gentlemen who now devote themselves to work in pathological laboratories and no longer visit the wards, had been compelled to study for three months in a children's hospital, we should have been spared such phrases as "rheumatism—that is, arthritis," and it would never have occurred to anyone to describe as an "attenuated pyæmia" a malady which is as definite and distinctive, and as certainly microbic, as pneumonia or typhoid, which is one of the most virulent diseases of childhood, and which never produces pus.

We must give up the conception of acute rheumatism as a form of arthritis of adults, with occasional metastasis to the heart, and with certain "peculiarities" when it occurs in childhood. We must insist on the conception that it is a microbic toxæmia most virulent in early life in which the heart is invariably affected to a greater or less degree, but the joints slightly and often not at all, with the peculiarity that when it occurs in adults the most prominent symptom is often a painful arthritis. It was from this point of view that I wrote the article on Acute and Subacute Rheumatism in the first volume of Dr. Allchin's "Manual of Medicine" a few months before the epoch-making discovery of the rheumatic diplococcus by Poynton and Paine, and subsequent experience has only confirmed the conviction that this is the right way of presenting the facts.

If we are to seek for an improvement in the treatment of acute rheumatism, it must surely be based on the great and universally acknowledged curative effect of sodium salicylate in cases of rheumatic arthritis. This drug is not a cure for all kinds of arthritis; the remarkable results obtained by it in rheumatic arthritis are not observed if it be used for a tuberculous, pneumococcal, or gonorrhœal joint-inflammation. The prompt subsidence of a rheumatic arthritis under its use can only be due to a definite antagonism to the rheumatic process or microbe—in other words, its action is specific. The general unwillingness of physicians to admit this really unavoidable conclusion is due to

the fact that the doses given are usually so small that only the more easily checked manifestations of acute rheumatism—the arthritis and the pyrexia—are fairly controlled. But if the inference is sound, may it not be possible, by a considerable increase of the dose, to produce a definite arrest of the disease in place of the imperfect control exerted by the small doses given at present? The question seems reasonable, but the possibility of an affirmative reply must depend upon the successful avoidance of the unpleasant symptoms which salicylate is apt to produce. Fortunately, these are much less troublesome in children than in adults; indeed, among the toxic symptoms observed in adults vomiting is almost the only one which gives trouble in childhood.

The addition of sodium bicarbonate to each dose of the salicylate, in an amount always double of the amount of the salicylate, is a very effectual means of diminishing these side-effects, and the unpleasant taste may be covered by glycerine and peppermint water or chloroform water. The use of sodium bicarbonate in combination with the salicylate has two additional advantages. It helps to neutralize the acid toxins of the microbe which Dr. Ainley Walker and Mr. Ryffel found, by special culture of the diplococcus, to be partly formic and partly acetic acids. And when we remember Dr. Gaskell's discovery, related in the third volume of the *Journal of Physiology*, that dilute lactic acid causes dilatation of the isolated frog's ventricle and finally arrest in diastole, while dilute sodium hydrate causes increased contraction with diminishing size until the ventricle stops in systole with its cavity reduced to *nil*, we may be reasonably sure that in man the administration of large doses of sodium bicarbonate must tend to cause a lessening of the pernicious cardiac dilatation of acute rheumatism.

Potassium salts should not be used, for the potassium base is really a cardiac depressant.

It is desirable to administer the combined salicylate and bicarbonate in frequent, but at first in moderate, doses. If the frequency be every two hours from 6 a.m. to 10 p.m., with one additional dose during the night, the total daily dose of each drug will be ten times the amount of the single dose. The amount of each should be increased daily, or every second day, until any unpleasant side-effect, such as vomiting, deafness, tinnitus, vertigo, or a tendency to delirium, is observed. In children, drowsiness, an acetone odour of the breath, acetonuria, and a slowing and deepening of the respiration must always be looked out for; if neglected, these may lead to "air-hunger" and fatal coma. Such symptoms are specially liable to occur if the child is costive, and may

then be produced by quite small doses of salicylate, especially if the urine is too acid. *They may be entirely prevented* by careful relief of constipation, conjoined with the administration of a sufficient amount of sodium bicarbonate. Enough must be given to render the urine alkaline, and a surprising amount is often needed to effect this in a rheumatic child. Twice as much as the dose of salicylate is usually sufficient, but occasionally in young children it is not quite sufficient, and extra doses of the bicarbonate alone are necessary to make the urine alkaline. It is also a good rule, suggested by Dr. Reginald Miller, Medical Registrar at the Hospital for Sick Children, never to increase the dose of the salicylate unless the patient's bowels have acted on that particular day.

If, through neglect of these precautions, any of the above-mentioned symptoms of acid poisoning occur, it may be necessary to omit the salicylate altogether, and to increase the amount of the bicarbonate—even to a drachm hourly. With these precautions the administration of large doses of salicylate is quite safe. The only fatal case in my own experience occurred seven years ago, in a child to whom the salicylate was given *without any bicarbonate* and who was costive. The dose of salicylate was very small, only 20 gr. daily (four 5 gr. doses), but by an oversight it had been continued for ten weeks, and the importance of the prevention of constipation was not then recognized. The credit of first pointing out the danger of constipation in children taking salicylate belongs to Dr. Langmead, formerly Medical Registrar at Great Ormond Street, who published a valuable paper in the *Lancet* for June 30, 1906.

As soon as any unpleasant symptom due to salicylate occurs in an adult, or if vomiting occurs in a child, the administration of the drug should be suspended for a few hours. Two, or three, doses may be omitted; even a twelve-hour interval may be allowed. But the elimination of salicylate in the urine is rapid, and the interval should not be long. After a few hours the medicine should again be given, but in reduced amount, one-half or two-thirds, according to the intensity of the symptoms observed. In many cases even the same dose as before will not produce a recurrence of the toxic effects, and I have sometimes suspected that they may be due in part to a bactericidal influence of the drug, destroying a large number of diplococci so quickly that their internal toxins are thrown in large amounts into the circulation. But it is wiser to reduce the dose of the remedy when it is again given, and to increase it the next day and continue to increase it as before. Almost always it is found possible in this way to reach a much larger dose than caused toxic symptoms at first, sometimes even three or four times

as much. If in the course of this increase of dose any fresh toxic symptom occurs, the same plan of arrest and temporary reduction should be adopted as before. There are very few patients who cannot be trained in this way to tolerate large amounts of sodium salicylate, and there is really no need for any of the German substitutes. But one must expect to find every now and then a patient who is specially susceptible to this drug, as others are to mercury, quinine, iodide, or bromide, or even to articles of diet such as eggs.

The initial dose of sodium salicylate may be: for an adult, 15 gr., or 150 gr. daily; for a child aged 7 to 12, 10 gr. or 100 gr. daily; for a child aged under 7, 5 gr., or 50 gr. daily, given in each case with twice the amount of sodium bicarbonate. The daily increase of dose may with advantage be from 2 gr. to 5 gr. in the individual dose—that is, from 20 gr. to 50 gr. in the total amount given daily. The increase in the dose should be progressive until the evening temperature is and remains normal. The amount needed for a child may be nearly as great as for an adult, for in a child the infection is more virulent and renal elimination is more active. In some mild cases of acute and subacute rheumatism it may be sufficient to increase the dose of salicylate to 150 gr. daily, but in many it is desirable to increase it to 200 gr. or 250 gr., and in some severe cases to 350 gr. or 400 gr. Chorea often requires 250 gr. to 350 gr., or even 400 gr. The doses usually given in this disease are much too small; large doses often cause rapid improvement. But in chorea time is often a necessary factor in the cure; damaged cortical cells require time for the restoration of their nutritive equilibrium after the arrest of the deleterious action of the rheumatic diplococci. A chorea treated early is often promptly arrested by large doses of sodium salicylate and bicarbonate, but a case which is already chronic will require a longer time.

In the chronic rheumatism of adults I have seen improvement commence when the daily dose reached 450 gr. or even 500 gr., while small doses may produce no apparent effect.

The maximum dose in my own experience was given to a boy, aged 15, with severe pericarditis, endocarditis, a much dilated heart, and a tremendous crop of subcutaneous and subperiosteal nodules, many of large size. In this case the dose was raised without any difficulty, and quite rapidly, to a maximum of 600 gr. of salicylate with 1,200 gr. of bicarbonate daily. This dose was given for two days without any ill effect. After a day free from medicine, it was resumed at the lower rate of 500 gr. of salicylate and 1,000 gr. of bicarbonate, given for six

days in each of the three following weeks. This was followed by a gradual reduction. An icebag was also applied over the heart for the cure of the pericarditis. The final result was most satisfactory.

What are the observed effects of these larger doses of sodium salicylate in acute and subacute rheumatism? Rapid relief of pain and subsidence of arthritis; fall of temperature to the normal, with little or no tendency to subsequent rise; rarity of relapse; marked diminution of the torpidity and depressed look of the rheumatic child; and as regards the heart, an easily observed reduction in size of the dilated left ventricle, while the cardiac impulse becomes stronger and more localized. It would be unreasonable to expect that a murmur due to endocarditis should immediately disappear, but the improvement in the condition of the muscular wall of the heart is often most striking. The rheumatic nodule is specially resistant to treatment, for its abundance of fibrous tissue tends to hinder a bactericide from reaching the diplococci which it contains. Yet under the influence of large doses of salicylate and bicarbonate even large nodules, such as were at one time said to be "equivalent to a sentence of death," will melt away. In the boy already mentioned as many as 216 nodules, some of large size, were counted on one day; yet when he left the hospital not a vestige of them remained.

Is it possible to assist the curative influence on the heart by the use of any other therapeutical measure? The answer to this question is very decidedly in the affirmative. In rheumatic pericarditis the extraordinary value of the local application of ice is one of the most certain facts in therapeutics. I advocated the use of the icebag in pericarditis at the Nottingham meeting of the British Medical Association in 1892, and I have used it for the treatment of this condition ever since. Its curative influence is very great. But in using it two points must always be borne in mind. The patient's lower extremities must be kept constantly warm by hot-water bottles, and any considerable dilatation of the right auricle must be relieved by leeches before the ice is applied—the indications for leeches being an extension of the cardiac dullness in the fourth right intercostal space to two finger-breadths, rapid respiration, restlessness, and some degree of cyanosis of the lips and face.

The beneficial action of the icebag in pericarditis is clearly a local one, and it suggests the probability that this measure may be useful also in rheumatic myocarditis and possibly even in endocarditis. I think there is no doubt that the application of an icebag to a dilated rheumatic heart (with the precautions already mentioned) certainly causes a more rapid reduction in size of the left ventricle and an increase

in strength of the cardiac impulse. And in some cases in which evidence of endocarditis has been present, I have observed under its influence a notable retrogression of the auscultatory signs. The earliest indication of an endocarditis is usually a systolic apex murmur, to which a doubling of the second sound at the apex is often soon added, and in some cases the second element of the double second sound becomes changed into a short mid-diastolic murmur. Under the local influence of ice I have noticed first the disappearance of the mid-diastolic murmur, and the reappearance of the double second sound, soon followed by the disappearance of the doubling, leaving only a systolic murmur and a second sound, and at a later stage the systolic murmur has tended to disappear.

I have gradually made more frequent use of ice in the treatment of cardiac rheumatism apart from pericarditis, and can recommend it as certainly helpful. But the two precautions already mentioned must be always remembered. The combination of ice to the heart with the use of large doses of sodium salicylate and bicarbonate has yielded very remarkable results in practice, and I speak from the experience of careful trials carried on for three or four years in a hospital service of sixty beds, consisting of twenty-five beds at the Hospital for Sick Children, with five beds for children and thirty for adults at St. Mary's Hospital.

My conviction is that if these methods were adopted universally, the lives of many rheumatic children would be saved, and that an enormous reduction would be effected in the prevalence of rheumatic heart disease in the adults of the next generation. How much suffering would thereby be prevented can be readily imagined by those who have watched the miserable downward progress of cases of chronic heart disease, the result of former rheumatism.

I commend this method of treatment to your favourable consideration. I submit no statistics, for statistics presented by the advocate of some special treatment are justly suspected as tainted by an involuntary bias. But I ask you to try the method for yourselves. And if to some there seems to be danger in these large doses, I would beg you to remember that an excessive timidity in dosage, due to the fear of doing harm, may often be more detrimental to the patient than a cautious boldness. In medicine a sin of omission is more lightly regarded than a sin of commission, *but it may be even more disastrous*. In advocating a bolder course I have endeavoured to state clearly the precautions which are necessary. Let no one adopt the former and yet neglect the latter.

DISCUSSION.

The PRESIDENT (Dr. Burton Brown) thanked both the authors for their interesting papers. For his own part he could say very little, as he left England before the salicylic acid treatment was known, and in India acute rheumatism was very seldom seen, which was remarkable, seeing what sudden changes in temperature were experienced there. He had gone out before sunrise with the temperature at 35° F., while at noon it was 90° F. He had also gone out in his brougham in a temperature of 110° F., and then entered a room at 65° F., or got into a bath with a temperature of 60° F. When he went out, the idea was that rheumatism was caused by a chill, but his experience in India seemed to disprove that.

Dr. W. J. MIDELTON agreed that there was a diplococcus which was responsible for acute rheumatism, that it persisted, and that once a person had the disease he never got rid of the organism. He thought salicylate of soda neutralized the toxins rather than that it killed the organisms. For a long time he had given salicylate of soda in influenza for its diaphoretic effect. Dr. Stockman asked for another drug other than salicylate; he, Dr. Midelton, thought cantharides had been too much neglected in this respect: it could not very well be given by the mouth, but it could be applied externally in various ways. He had experimented with it on himself, and could now show a rash on his chest produced by the drug. For chorea, he thought "chlorestone" (?), manufactured by Parke, Davis, was a powerful drug. He thought the previous history of the patient was of great use in diagnosing rheumatic conditions. In one case now under his care he thought the influenza bacillus was responsible for the condition, and in another that of scarlatina. He had been surprised that no mention had been made of treatment of the nervous system in connexion with the disease, yet that was very important. One could not very well give many drugs, but counter-irritants could be applied. In the case due to influenza, in which the symptoms resembled those of rheumatic fever, he directed a mustard plaster to be applied alternately over the cervical enlargement of the spinal cord and over the lumbar enlargement. He gave opium at the same time—a much neglected drug. The pain in the arms and legs in this case was very great, but rapidly subsided under this treatment and did not return.

Dr. F. J. POYNTON desired to protest against the way in which Professor Stockman presented the bacteriology of rheumatic fever. He spoke of Achalmé's bacillus, the diplococci, the staphylococci, and possible protozoa as of equal importance, and he, Dr. Poynton, maintained that, scientifically, that was not the correct interpretation of the present position. No doubt Dr. Stockman was well acquainted with the original papers of Achalmé, and he would remember that authority never succeeded in producing anything like rheumatic fever in animals, though he caused bleeding and œdema, apparently a condition of septic poisoning. The diplococcus was a streptococcus, but the question was what sort of streptococcus. No one to his knowledge had shown that any

staphylococcus could produce rheumatic fever experimentally. What was said about a protozoon was a pretty theory, but it lacked proof, for no scientific point had been put forward in favour of it. But with regard to the diplococcus, anyone could see from the specimens in the College of Surgeons' Museum that endocarditis, pericarditis, and arthritis could be caused by it experimentally. Therefore, there was no doubt it stood well ahead of all other organisms in respect to the cause to date; and all teachers of medicine, he claimed, should put the cause of rheumatic fever and what had been done on the subject in its correct perspective. With regard to treatment, he was interested in Dr. Stockman's remarks as to the difference between the cases when the disease was in the fibrous tissues, causing oedema there, and those where arthritic effusion was caused. There certainly was a striking difference, but that did not prove that a different organism was at work. When an organism set up arthritis it got into the connective tissue of the joints; why should it be considered a different organism when it was active in the connective tissue of the fascias?—a difference of virulence. The position of salicylate of soda was one of very great interest, but he could not see that its specific character had been proved. One needed, he thought, the same attitude of mind concerning salicylate as about all strong drugs in all diseases which were not thoroughly understood; in some cases it acted, and in others it did not; and it was not yet known why it did and why it did not. Neither was it known how much rheumatism was due to the bacteria, to the poison of the bacteria, and the peculiar tissues of the patient. One, in fact, relied on clinical experience, and he awaited with considerable interest the results of those who followed Dr. Lees's lead and studied the effects of large doses and those who preferred smaller doses, or who gave large doses in some cases and smaller doses in others. He strongly supported the idea of giving a purgative before commencing the salicylate treatment. That was pointed out to him fifteen years ago by a Sister in the Victoria Ward of St. Mary's Hospital.

Dr. W. H. WILLCOX expressed his high appreciation of the valuable papers which had been heard. He had been for many years associated with Dr. Lees in his work at St. Mary's Hospital, and he had also given some little assistance in the well-known work of Drs. Poynton and Paine. It had been stated by Professor Stockman that bacteriologists said the rheumatic diplococcus was a terminal infection, that people who had had rheumatism badly, during the last few hours of life, had their blood and body generally flooded by organisms from somewhere, and that the diplococcus in question was the last organism to find its way into the tissues in that way. But that was not true, because one of the first patients from whom the organism was isolated was one under the care of Dr. Lees—a boy, aged 18, who was employed at Whiteley's stores. The boy had acute pericarditis, and leeches were advised, but Dr. Lees gave the speaker permission to perform venesection instead. Drs. Poynton and Paine were present, and strict antiseptic precautions were observed. The blood was run into broth, and a very valuable strain of diplococci was obtained. Subcultures of the organism produced all the symptoms of rheumatism in animals. Only

last year he had a letter from a young man in Wales expressing his great gratitude for that operation, which he felt sure had saved his life: he had had no illness since. It was the same person. There were many other points of great interest. He could thoroughly endorse all that Dr. Lees had said with regard to large doses of salicylate: he had himself given as large doses to patients as Dr. Lees mentioned. He was sure that salicylates acted very much as did iodide. If a patient with a little cough were put on 3 gr. of iodide he often would get bad iodism, but if the dose were increased to 20 gr. the symptoms of iodism would disappear. The drug, then, seemed to have a diuretic effect, and it was more rapidly excreted. Often small doses of salicylates would cause deafness and dizziness, whereas large doses would not. There was one point in connexion with salicylate which was not generally known. Professor Stockman said the drug was excreted with rapidity: the speaker had given 100 gr. of salicylate of soda to a patient, and four or five hours later there was no trace of it in the urine; that is, no reaction with ferric chloride. That was important, because in acute cases salicylate of soda was indicated, whereas when a more prolonged effect was needed a drug with a slower action should be employed. If 10 gr. of aspirin were given it would be found in the urine for two days as salicyl-uric, and there was a purple reaction with a solution of ferric chloride. The poisonous effects of salicylic acid were not often seen, but seemed more likely to occur where there was constipation. He had the opportunity of examining on the chemical side the cases published by Dr. Langmead to which Dr. Lees referred. There was one useful clinical test—namely, the test for the presence of acetone in the urine; if the person had symptoms of salicylic acid poisoning there was not only a smell of acetone in the breath, which was very deceptive, but acetone was present in the urine, and if the urine were mixed with a solution of sodium nitro-prusside, and a solution of strong ammonia carefully poured above the mixture, the junction of the two fluids was marked off by a line of rose-violet colour which took two or three minutes to develop. A few months ago he was asked to see a case of rheumatism with alleged salicylic acid poisoning. The patient was delirious, with a temperature of 105° F. and rising, and there was said to have been deafness. He tested the urine, but there was no acetone, the pupils were widely dilated, and on referring to the medicine chart he found a large dose of hyoscyne had been given a few hours previously, because the patient had not been sleeping well and was delirious. He did not stop the salicylate of soda, but trebled the dose, and the patient immediately got better. He would not have taken such a strong line if he had not found that there was no acetone in the urine. Dr. Lees had referred to the beneficial effect of alkalies, such as bicarbonate of soda, and he agreed with him. But physicians often lost sight of the fact, in their prescriptions, that the maximum solubility was about 1 in 16, so that if 1 dr. of bicarbonate of soda were prescribed for a dose, it should be given with 2 oz. or 3 oz. of water; otherwise the patient would be getting less than a dose in the earlier portions and a much concentrated one later. In the alkaline treatment of the disease it should be remembered that

the citrates were often absorbed much better than were the bicarbonates, and had a more powerful alkaline action. If bicarbonate of soda were given, a large quantity of it was turned into common salt in the stomach.

Dr. J. GRAY DUNCANSON asked whether Dr. Stockman had made any observations as to the effect of salicylates on the leucocytes. He had recently observed in an American text-book on pharmacology (Sollmann) the statement that "the leucocytes of the blood are doubled an hour after taking salicylates; but return to normal within two hours," which might explain the benefit of their administration at short intervals. With regard to chills, whether rheumatic or influenzal, he thought the simplest plan was to indulge in a little polypharmacy and prescribe the salicylate of soda with ammoniated tincture of quinine and sodium bicarbonate. In connexion with the cases mentioned by Dr. Lees of rheumatism complicated with heart trouble, he thought the great secret was to combine the treatment with a long period of absolute rest, as recommended by Sir Lauder Brunton, care being taken to spare the heart an unnecessary beat. That point, he was sure, had not been sufficiently insisted on in the past.

Dr. H. C. CAMERON said that Dr. Lees advocated the use of larger doses of salicylate so persuasively that one could hardly help being convinced, but he would like to know what evidence there was that such large doses were really absorbed. He was not aware of any work undertaken to find out the limit of absorption of sodium salicylate. He thought one explanation of the freedom from salicylate poisoning of patients who were taking large doses might be that there was little more of the drug actually absorbed than if the dose had been much smaller. He did not know what was Dr. Lees's explanation of the dangerous symptoms from the drug in those who suffered from constipation—a sufficiently common clinical complication. Possibly such patients were really absorbing more than those who were taking larger doses, but whose bowels had previously been properly opened. He had noticed in the routine examinations of urine in the wards that albuminuria was not uncommon in those who were undergoing treatment by salicylates. There were also several cases published in the Guy's Hospital reports, some time ago, by Dr. Shaw, in which hæmaturia had been a complication. Dr. Lees did not say whether in carrying out the treatment he had noticed any symptoms pointing to involvement of the kidney.

Professor DIXON said it was not often that at a therapeutical meeting precise and definite evidence was brought forward, but that day they had had definite evidence of the action of salicylates: not simply a physician's impression, based on his practice. Clinical observation involved the personal factor, and as such had little scientific value. At that meeting, however, the evidence was perfectly clear that a definite increase in dosage produced a certain definite effect: the evidence was as valid as if it had been the outcome of the laboratory—of experiments performed under precise conditions—and he thought Professor Stockman should be congratulated for having produced such a communication. Professor Stockman mentioned salicyluric acid, and he had kindly provided him,

Dr. Dixon, with some, which he had prepared from the urine of his patients. He took 30 gr. of it without the slightest effect, and he administered large doses of it to cats, under anæsthesia, but no action was observed; it did not lower the blood-pressure, even as a large injection of salicylates, thrown into the circulation, would do. He had been rather surprised to hear the views which had been expressed on the bacteriology of rheumatic fever; he had seen streptococci and staphylococci cause vegetations on the valves of the heart and joint-troubles in rabbits; he was sorry there were no bacteriologists present to give the modern view. He strongly held to the view which Professor Stockman mentioned at first: that no organism had yet been isolated which could be regarded as the cause of acute rheumatism.

Professor STOCKMAN, in reply, reminded his hearers that before he commenced his paper he said he had not come to go into matters of detail which were known to all members equally with himself. He wanted rather to touch on matters about which people were not agreed. That was where meetings were useful. He did not intend in any sense to depreciate Dr. Poynton's work in connexion with the bacteriology of rheumatic fever: he had only time to touch on that aspect in a short way; he was not himself a bacteriologist. He was aware of Dr. Poynton's work, and that gentleman had always sent him his papers, which he had read repeatedly, and some of them only a week ago. He had himself examined, and also with the assistance of skilled bacteriologists, a number of patients, but he had never been able, out of the joint-effusion or out of the blood, to grow the bacterium. And he noticed that Vidal said he had examined the blood of fifteen rheumatic fever patients, and also their joint-fluid, without finding an organism; yet he had used all kinds of media, and had the resources of the Pasteur Institute at his back. He obtained from Drs. Poynton and Paine some of their material, and again, with skilled help, had tried to infect rabbits with it, but unsuccessfully. From Dr. Beattie, of Sheffield, a growth of the organism had also been obtained, and again there had been lack of success in inoculating rabbits. It might be a fault in the technique or in dosage, although they had given large doses. He had said it was very difficult to distinguish pyæmia from rheumatism, and he still thought so. There was need of a definite diagnostic, as for tubercle or typhoid. It should be possible to work out scientific questions in a scientific way without any idea that an observer was being slighted. In answer to Dr. Duncanson, he had not observed leucocytosis in healthy people after salicylates, but in rheumatic cases it occurred, though he did not know whether that was due to the salicylates. He thanked those members who had joined in the discussion.

Dr. LEES, in reply, desired to express his appreciation of Professor Stockman's paper, and especially of the clear statement that acute rheumatism is a definite fever, and that sodium salicylate has a specific curative effect. The charts exhibited by Professor Stockman showed very convincingly the prompt antirheumatic action of this drug when given in sufficient doses, and the fact that larger doses would sometimes accomplish at once a result which

smaller doses had failed to secure. The experimental demonstration that in rabbits large doses of sodium salicylate had no depressing effect on the heart was also of great value, and confirmed his own observations in cases of rheumatism. With regard to the bacteriological controversy, it seemed to him that one positive observation outweighed many negative ones, which might be due merely to slight (perhaps very slight) differences in the precise method adopted. Two points of evidence within his own knowledge seemed to him very important. The first was the fact that a rheumatic nodule of recent origin in a child in St. Mary's Hospital, when excised by Dr. Poynton under aseptic precautions, at once placed in a nutritive medium and incubated for forty-eight hours, showed on section (as he could testify) an exuberant growth of a diplococcus in pure culture. Since the nodule is especially distinctive of rheumatism, this observation appeared to him very convincing. The second fact was (as stated by Dr. Willcox) that a diplococcus had been obtained from the blood of more than one of his rheumatic patients during life, and that in one of these cases the infection was certainly not "terminal," for the patient is still alive and in good health eight years after the venesection. With regard to the administration of sodium salicylate he wished to add two statements in confirmation of what he had already said. His colleague, Mr. Leslie Paton, told him—and he had Mr. Paton's permission to repeat it at that meeting—that he had used the method of administration advocated in this paper (and previously) in cases of rheumatic iritis with great success. Mr. Paton mentioned especially the case of a gentleman who had been very susceptible to sodium salicylate, so that he had poisonous symptoms after 5 gr. doses; but by adopting the method described the dose was increased until he was taking 25 gr. every three hours, without difficulty, and with very gratifying success. A second confirmatory statement was found in a paper in the *Western Canada Medical Journal*, sent to him by a former house-physician at Great Ormond Street now practising in Vancouver, who had written a paper on the prevention and treatment of some common diseases of childhood. In that article he found the following passage: "I have seen so many cases of chorea treated by salicylate of soda and alkalies in hospital wards, and compared the rapidity of their recovery and absence of heart complications with those treated by arsenic or simple rest, that I am confident it is the best line of treatment." That was quite independent testimony in confirmation of what he had advanced.

Therapeutical and Pharmacological Section.

January 5, 1909.

Dr. T. E. BURTON BROWN, C.I.E., President of the Section, in the Chair.

Tissue Antisepsis with Reference to Animal Infections.

By A. R. CUSHNY, M.D., F.R.S.

SOME thirty years ago, when the bacterial origin of surgical infections became generally recognized, and Lister had shown how those organisms could be held in check by means of drugs, it occurred at once to many minds that some or all of the acute fevers might be the result of similar organisms, and high hopes were entertained that the ingestion of antiseptics might cure all these diseases by destroying the organisms in the tissue. In support of such hopes, the successful treatment of malaria with quinine and of syphilis with mercury were cited. Accordingly we find in the medical Press reports of the results of carbolic acid, corrosive sublimate and other antiseptics in the treatment of the acute fevers, such as scarlet fever, pneumonia, or measles, and, above all, in the various forms of tuberculous disease. And, as a matter of fact, this idea did lead to the use of salicylates in rheumatism, and I was glad to hear Professor Stockman place this drug among the specifics with mercury and quinine. The hopes that a drug would be found to act as a general bactericide in the tissues have, however, receded as our knowledge of the enemy has increased. We know now that the microbes are very much more tenacious of life than the higher organisms, that their antiquity, transcending that of man by many millions of years, has permitted a much more perfect adaptation to their environment, and that only those have survived which are capable of meeting a much greater number of untoward circumstances than the human tissues. Among those untoward circumstances are drugs. The mammalian

tissues are much more susceptible to drugs than any of the microbes. This is obvious to anyone who considers the fatal dose of almost any drug and compares it with its antiseptic action. Take, for example, corrosive sublimate, and suppose that in a solution of 1 per 5,000 it is absolutely fatal to microbes, though this is a large over-statement. In order to disinfect a bulk of fluid corresponding to a man of 10 st. weight, we would have to add over 200 gr. For organ antisepsis some body must be found which is very much more poisonous to microbes than to man. And the micro-organisms differ so much from each other that it is hardly to be hoped that any such drug exists. The idea of a general disinfectant for the tissues has, therefore, been abandoned by most thinking minds,¹ but hope springs continual in some breasts, and one still hears of audacious attempts to introduce the millennium in this way.

A few years ago the silver compounds were the favourite, and one heard of the disinfection of the tissues by the intravenous injection of colloidal silver, and several gynæcologists satisfied themselves that they were in the van of scientific progress when they rubbed colloidal silver into the skin of patients suffering from puerperal infection. Still more recently the medical Press chronicled the treatment of septicæmia by the injection of formaldehyde solution into the veins. I wonder if the author had ever witnessed the hardening of tissues under formaldehyde which is used in histological research, or considered the relative resistance of the human tissues and the microbes to formaldehyde. The keynote in this matter is that the disinfection of a surgical infected wound is only possible at the expense of the tissues surrounding it. We can destroy the microbes in a wound, but only by injuring the cells with which they are in contact. In some local internal infections the idea of disinfection may still be entertained, therefore, if it is possible to risk some injury to the cells of the host surrounding the organisms. For example, in the alimentary canal disinfection may be possible, though the results hitherto have not been very striking, and the general feeling is that the emptying of the canal (that is, the aseptic procedure) is the more satisfactory. But the organ in which antisepsis is chiefly sought at present is the lung. It seems plausible enough that if an antiseptic be inhaled, it may act on the organisms in the lung; and if the lungs were leather bellows, disinfection by inhalation would be a natural and

¹ It has, in addition, been shown recently that some bodies which are efficient bactericides in the test tube became very much less active when they have to act in the presence of the colloids of the blood and tissues (Bechhold, *Zeitschr. f. physiol. Chem.*, Strassb., 1907, lii, p. 177).

highly efficacious means of treatment. But the pulmonary epithelium is a very delicate substance, injured by the slightest variation in temperature, even by such a harmless gas as pure oxygen, and would certainly be killed long before the hardy tubercle bacillus. Fortunately, attempts at disinfection by inhalation are frustrated by the reflexes of the respiratory passages. Injurious substances are excluded by coughing and closure of the glottis. But one still hears of ozone treatment. Bournemouth still celebrates the virtues of its pines in providing disinfectant breezes, and fortunes are still made by the judicious appreciations of the value of various sprays and inhalers which are supposed to carry drugs to kill the bacillus while sparing the lungs in which it lives. The creosote treatment must be mentioned here—the ingenuous hope that the traces which are eliminated by the lungs can affect the microbes—but I need hardly enter on the discussion. Creosote treatment is as useless as the other methods adopted.

The searcher for specifics in the treatment of infectious diseases has hitherto been buoyed up by the success which has attended the use of the two great specifics, mercury and quinine. But within the last few years investigation has shown that syphilis and malaria are not to be classed with the ordinary infectious diseases of which the cause is known, since the organisms of malaria and syphilis are animal, and differ in essential features from the causes of tuberculosis, diphtheria, pneumonia, and the various forms of suppurative disease. One of the characteristics of these animal infections is that the organism is much more amenable to treatment than the vegetable organism. They do not seem to have adapted themselves so successfully to their environment, and perhaps all these diseases are of more recent date than those originated by the bacterial invaders. In addition, the animal parasites do not seem to secrete toxins, at any rate, to the same extent as the bacteria, and in consequence the host does not meet them with the formation of antitoxins. The latest of these animal infections to be discovered is the sleeping sickness of tropical Africa, which was soon found to be due to a minute parasite, the *Trypanosoma gambiense*, and almost immediately this disease was found to react to various specifics. The first of these was arsenic, which has been used in the inorganic preparations, of which Fowler's solution may be taken as the type, and also in organic combinations such as atoxyl or sodium-aminophenyl arsenate. A certain confusion has arisen from the introduction of this preparation, which, according to most investigators, appears to act more satisfactorily than the older form. Yet it is quite devoid of action on the

trypanosome outside the tissues, and can only become specific for it when it is changed in the body. The active therapeutic agent here is undoubtedly the arsenic content, but it is readily conceivable that atoxyl may possess some advantage over the inorganic preparations in virtue of its physical characters. This may be made intelligible by comparing it with such a couple as ethyl alcohol and acetic acid. Alcohol possesses certain effects on the central nervous system in virtue of its power of penetrating the nerve-cells, while acetic acid, or the acetate, is unable to do so, and remains practically devoid of effect. In the same way, atoxyl may be able to penetrate into tissues from which inorganic arsenic is excluded, and may there free arsenic. Much of the atoxyl administered passes through the body unchanged, and is valueless as a disinfectant, but the small proportion which does not thus escape is capable of destroying a large proportion of the parasites.

Arsenic is chemically one of the series, nitrogen, phosphorus, arsenic, antimony and bismuth, and its neighbours phosphorus and antimony have always been recognized as possessing somewhat similar pharmacological properties. When arsenic proved to have specific trypanocidal properties, the next step was to investigate antimony, and a preparation which was tried on rats at my suggestion was found to destroy the trypanosomes in the blood even more rapidly than arsenic. A good deal of difficulty is met with in avoiding the irritant and caustic effects of antimony, which are much more developed than those of arsenic, but apart from these the antimony treatment appears to be a distinct advance on that with arsenic. The next member in the series, bismuth, has also been tried in trypanosomiasis, but proved to be too poisonous to the host to be available, though it also is destructive to the parasites. There can, therefore, be no doubt that in the arsenic, antimony, bismuth series we have a further example of specific action which may be compared with that of quinine in malaria and mercury and iodides in syphilis. That is, these specifics are much more poisonous to the animal parasites which cause these diseases than to the hosts which harbour them. The further curious fact emerges, namely, that these specific drugs are probably more poisonous to those parasites than to other lower organisms. Thus the trypanosomes in a rat of 200 grm. weight may be destroyed by about 1 mg. of antimony. Assuming that all the antimony is in solution in the tissues of the rat, this means that 1 part of antimony in 200,000 is sufficient to destroy the trypanosome.¹ About the same amount of

¹ In man, Broden and Rodhain found 0.1 gr. of tartar emetic injected intravenously sufficed to clear the blood of the patient suffering from sleeping sickness. This would correspond to about one part of antimony in two millions of body-weight.

arsenic also acts upon the trypanosomes in the rat, but the action is slower and apparently less complete, and approximately the same amount of bismuth acts on them, but the injury to the host is generally irreparable. We may, then, take it that this series is poisonous to the trypanosomes in the rat in the concentration of 1 : 200,000. The tissues, however, scarcely form the most favourable medium for the action of disinfectant substances, as Bechhold has recently shown, and one would expect that these poisons would be much more powerful in fluids containing less colloid. I have performed a number of experiments on the influence of these poisons on the common protozoa of the hay infusion, such as paramœcium and colpidium, and was surprised to find them living for days or weeks in comparatively strong solutions. The concentrations¹ necessary to kill these non-parasitic forms were:—

		Paramœcium and Colpidium		Trypanosomes in rat
Arsenic	...	1 : 3,400	...	1 : 200,000
Antimony	...	1 : 970	...	1 : 200,000
Bismuth	...	1 : 98	...	1 : 200,000

This group, therefore, acts very much more powerfully on the trypanosomes than on the harmless organisms of the hay infusion.² And the same holds true for quinine; for a dose of 15 gr. is often sufficient to destroy the malarial parasites in an ague patient—that is, about 1 part of quinine in 70,000 to 100,000, assuming that all the quinine is absorbed and in solution in the tissues at the same time. In some experiments with the colpidia of hay infusion I have found that these protozoa live in solutions of 1 : 10,000 of quinine for an indefinite time without any apparent diminution of their movements or of their rate of multiplication. The amount of mercury which is necessary to destroy the spirochæte in the tissues is not so definite, and I have, therefore, not ascertained its toxicity to organisms outside the body. A further anomaly may be pointed out; in the tissues the trypanosomes are destroyed at least as readily by antimony as by arsenic, and bismuth does not come much behind them. But the hay organisms are 3·6 times more resistant to antimony than to arsenic and tolerate 36 times as much bismuth.

¹ The concentration is calculated for the actual metals, not for the salts. In the experiments on paramœcium, &c., the metals were used in the form of the double tartrate of the metal and sodium.

² A further instance of the specific nature of these poisons is that moulds, as is well known, grow luxuriantly in 1 per cent. arsenic or antimony solutions, and these have very little action on bacteria.

In malaria and trypanosomiasis we have, then, parasites which are exceptionally sensitive to certain drugs—much more sensitive than ordinary protozoa. The extraordinary coincidence that these pathogenic protozoa are also highly susceptible to remedial agents might suggest that the drugs act not as direct poisons to the organisms, but by arousing some dormant activity in the tissues which destroys the parasites. If this were correct, however, one would expect that quinine would act as well in trypanosomiasis or syphilis as in malaria, whereas we know that it is valueless in these affections. And in addition these organisms are extraordinarily susceptible to their specifics outside the body. Thus, Löffler and Ruchs found the trypanosomes die in arsenic solution 1 : 200,000,¹ and Broden and Rodhain state that the *Trypanosoma gambiense* died in thirty-five minutes in a 1 : 500,000 solution of tartar emetic, which corresponds to one part of antimony in about one and a half millions. Even among the trypanosomes some are much more susceptible than others to antimony or arsenic—for example, the *Trypanosoma gambiense* is more susceptible to antimony than the *Trypanosoma lewisii*.

The study of the action of specifics has hitherto been hindered by the fact that the diseases in which they act have not been transmissible to animals, except with great difficulty. Now, since we are able to study the effects of specific drugs in the trypanosomiasis infections in animals, a more rapid progress in our knowledge of these drugs may be expected. One important point has already been made out—the rapid tolerance acquired for the trypanocidal drugs. When a rat infected with trypanosomes is treated with a dose of arsenic, the parasites rapidly disappear from the blood, and in a few cases never reappear. In the great majority of cases, however, one finds them reappearing in the course of ten to fifteen days, and on repeating the treatment they again disappear, but recur again, and this time the interval is shorter. And each time the interval in which the blood is free of the parasites becomes shorter, until finally they do not disappear at all, and the rat dies in spite of continued arsenic medication. The explanation is that, although most of the parasites are destroyed by the arsenic in the first instance, some few survive and propagate their kind, and this process goes on after each injection. One may consider that the parasites have acquired a new quality, as Ehrlich suggests, or, on the other hand, one may suppose that a very drastic selection of the fittest has taken place, only those naturally most resistant to arsenic surviving each time, until in course of

¹ Still more recently Ehrlich has stated that two arsenical compounds obtained by the reduction of atoxyl destroy trypanosomes *in vitro* in a dilution of one part in a million.

time a race is evolved which is indifferent to arsenic. This arsenic-resistant type may now be propagated from rat to rat a hundred times, a few individuals serving to propagate an infection in each animal, but the race remains arsenic-resistant, and animals infected with this race are not benefited by arsenic in the least.¹ Other trypanosome specifics act as usual, however, antimony destroying the arsenic-resistant strain about as readily as an ordinary strain. The practical inference to be drawn from these facts is that in treating trypanosomiasis one must strike hard at the beginning. If one begins with small doses, one not only does not destroy the parasite, but actually helps it to resist when stronger measures are taken.

Another point which is developing from this study is that it may be advisable to use not one drug, but as many as possible, in an infection. The recurrences in trypanosomiasis are due to some individuals which are more resistant to the drug than the average. Perhaps they have atavistic tendencies which give them the resistance observed in ordinary hay-infusion protozoa. If, however, two drugs are given at once it is unlikely that the same individuals will prove resistant to both of them, and an effective disinfection of the tissues may thus be possible. It is curious to find the most modern branch of medicine supporting the view that the exhibition of two drugs having apparently the same action may be more efficacious than either of them given alone in larger doses. The same view has long been held in regard to many other remedies in medicine, such as soporifics, anæsthetics, and, above all, purgatives. And the same combination of therapeutic measures is seen in the use of mercury and iodides in syphilis—another animal infection.

A further point which has recently emerged in trypanosoma treatment may be mentioned. When an atoxyl-resistant strain is obtained by treating a trypanosome infection in mice, the further treatment of infected mice with atoxyl has no further therapeutic effect. If now this strain be inoculated in a rat, it becomes susceptible to atoxyl treatment again, but again becomes resistant when retransferred to the mouse, even after passing through generations of rats. No explanation of this observation has been offered, and it is very desirable to have observations made of the reaction of atoxyl-resistant forms to arsenic outside the body. It may be suggested as an hypothesis that here again we have an interaction of two principles injurious to the trypanosome—one the atoxyl and the

¹ This has been termed a transmission of an acquired character, but may be equally well described as an example of natural selection, and the transmission is only in unicellular "immortal" organisms in any case.

other some antitrypanic body which may be called mouse-poison. In the mouse the trypanosomes become resistant to both of these. On transmitting these resistant forms to the rat they meet a new poison to which they are not resistant, and those that survive this and multiply are destroyed when they meet the combination of rat-poison and atoxyl.

Many other points of interest arise in this new method of therapeutic study, but I trust that I have made clear to you that a new study has been introduced, and one that promises to throw light on some very obscure points in general therapeutics.

Salicylates as Retentives : their Effects on Capillary Circulation, Blood-pressure and Uric Acid in the Blood.

By ALEXANDER HAIG, M.D.

Figures and Experiments by K. G. HAIG.

As I have pointed out in "Uric Acid," edition 7, chapter ii, the salicylates are solvents of uric acid, and, like the other well-known solvents (the alkalies, *i.e.*, alkaline salts of sodium and potassium), aid its elimination in the urine. And it is by this removal of uric acid that they relieve the acute arthritis of gout or rheumatism, as well as the chronic arthritis, myalgia and other irritation of fibrous tissue in any part of the body due to the same cause. But, as I also point out (previous reference), there are certain conditions in which salicylates cease to be solvents of uric acid; and in some of these conditions they may not only do no good in the above-named troubles, but may do actual harm, producing arthritis instead of removing it, and causing very decided rise of temperature and acute irritation of fibrous tissues.

Some of these conditions, about which much has been said already in "Uric Acid," are : the administration of salicylates in certain proportion with alkalies, or their administration in conditions (*e.g.*, heat and perspiration) which act like alkalies in increasing the alkalinity of the blood and decreasing the acidity of the urine.¹

¹ See also paper on the "Treatment of Bronchial Catarrh by Alkalies," *Brit. Med. Journ.*, 1908, i, p. 1100.

I have also referred to the well-known fact that salicylates are of comparatively little use in the arthritis met with in warm climates, or even in this climate in very warm weather; but I have shown that their activity can be increased under these conditions by keeping the patients cool and applying cold to their joints.

I have also shown that, within certain limits, acids aid the salicylates and increase their solvent power; this accounts for the fact that salicylic acid is a more powerful solvent than its sodium salt, also that aspirin and novaspirin are still more powerful, because in them an acid radicle has been combined with the salicylic acid. Salicylates also act best in fever when acidity of urine is high, and least well in conditions of debility where the acidity of the urine is low.¹

We have arrived, then, at this—that alkalies and heat, which diminish the acidity of the urine and increase the alkalinity of the blood, rather hinder the solvent powers of salicylates, while acids and cold, which have the opposite effect on the urine and blood, rather aid their solvent action.

In this paper I point out that salicylates, under certain conditions of dose and time, may be used to produce retention in place of solution and plus excretion; and that they will then not only do harm in arthritis, but may be used to do good in those collæmic conditions (headache, debility, depression, fatigue, &c.) which stand at the very opposite pole of causation from arthritis and are due to, and more or less directly associated with, the presence of excess of uric acid in the blood.

The results given in the figures were obtained by Mr. K. G. Haig working on his own blood and circulation in the alkaline tide hours. The capillary circulation being acted on by the capillary dynamometer² invented by me, and the rate of the return of colour (capillary reflux, or shortly, C.R.) being measured by a metronome beating half-seconds, the blood-granules and blood-pressure were measured by the methods and instruments described in "Uric Acid," chapter xvii.

The first thing to get is a standard of comparison, and this we have in fig. 1, which is a C.R. curve on a day when no drugs were taken, beginning at 7 a.m. and ending at 11.15 a.m. At 7 a.m. the C.R. was 7 half-seconds, the blood-pressure 130 mm., and the water 198 c.c. per half-hour. At 7.30 a.m., and from that to 8.45 a.m., the C.R. had slowed to 8 half-seconds, the water had fallen to 109 c.c., 20 c.c., and

¹ See "Salicylates in Diagnosis and Treatment," *Med. Record*, New York, December, 1907.

² Made by Hawksley, 357, Oxford Street, W.

17 c.c. per half-hour respectively, and the blood-pressure at 8.30 a.m. had risen to 145 mm. From 9 a.m. onwards there was a gradual quickening of C.R. till, at the end of the curve, it was only 6.4 half-seconds. Water remained rather scanty till breakfast was taken at 9.45 a.m., and then it rose a little, reaching 108 c.c. in the half-hour ending 11 a.m., when blood-pressure had fallen again to 130 mm. We

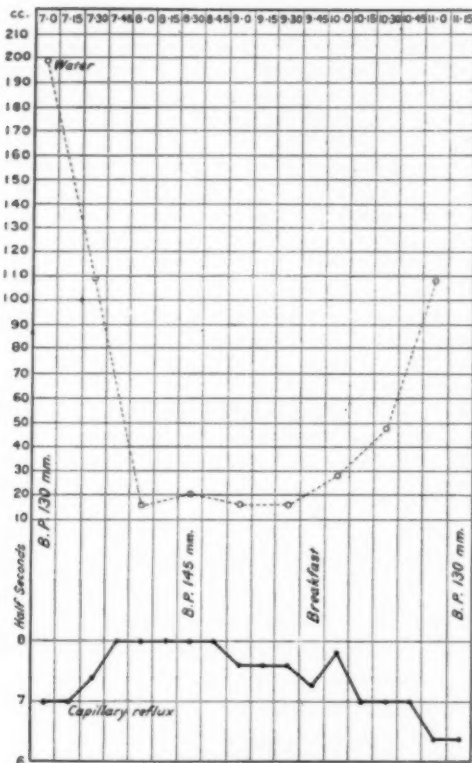


FIG. 1.

A day without drugs.

may note that the most scanty water went, as it generally does, with the slowest C.R., and that the water was more profuse before and after this with C.R. at 7 half-seconds and below. As usual, the blood-pressure rose with the slowing of the C.R.

Fig. 2 shows the effect of taking sodium salicylate 5 gr. at 9.30 a.m., when the C.R. was 7 half-seconds, and the proportion of granules to red cells in the blood was as 1 to 6. By 10 a.m. the C.R. had quickened to 5 half-seconds and the granules had diminished to 1 to 30, and at 10.30 a.m. the urine in an hour had run up to 300 c.c. From this time onwards there was a gradual slowing of C.R., until at 12.30 p.m. it had reached its original level at the beginning of the figure. We learn from this figure that the effect of taking 5 gr. of sodium salicylate was to

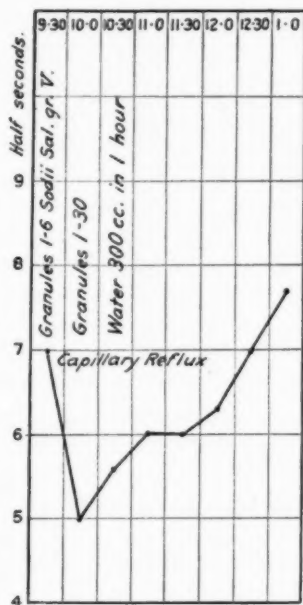


FIG. 2.

Effect of small dose of salicylate of sodium.

diminish the blood-granules, increase the urinary water and to quicken the C.R.

In fig. 3, $2\frac{1}{2}$ gr. of sodium salicylate were taken at 6.30 a.m., when the granules were 1 to 9, blood-pressure 130 mm., the water 66 c.c. per half-hour, and the C.R. 6.6 half-seconds. There was no decided quickening of the C.R.; on the contrary it slowed, till at 7.30 a.m. it was 8.4 half-seconds, and then the granules were 1 to 7, blood-pressure 150 mm., and water, which was 66 c.c. at the beginning of the figure,

had now fallen to 24 c.c. per half-hour. At 8.15 a.m. another $2\frac{1}{2}$ gr. of sodium salicylate were taken, and at 8.30 a.m. C.R. had quickened to 7.8 half-seconds and water had risen to 27 c.c.; at 9 a.m. C.R. had quickened to 6.2 half-seconds, granules had fallen to 1 to 29, and blood-

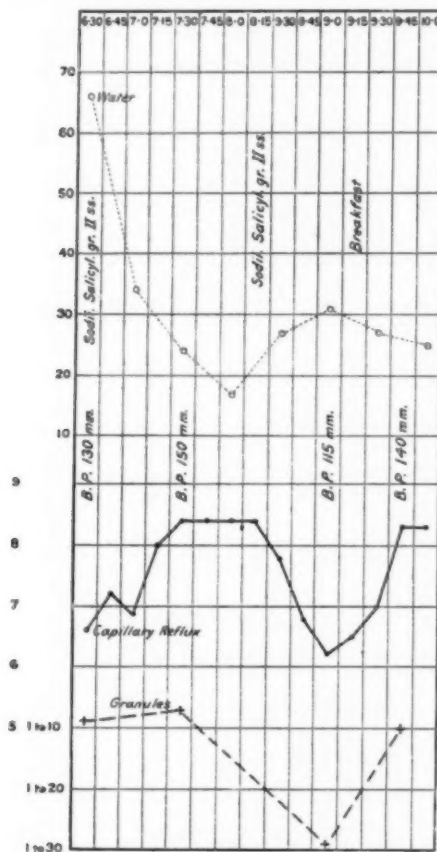


FIG. 3.

Effect of two small doses of salicylate of sodium.

pressure to 115 mm., while water had risen to 31 c.c. The rest of the figure merely shows the return of the reflux, and at 9.45 a.m. C.R. had slowed again to 8.3 half-seconds, granules were 1 to 11, and blood-

pressure 140 mm. We here see the value of C.R. as an index of the uric acid in the blood, as shown by the granules, the blood-pressure and the flow of urine, which are controlled by the capillary circulation and the things that affect it; and we here see that, as in fig. 2, the presence of a small quantity of salicylate clears the blood of uric acid and affects all the things that are dependent on this. If the C.R. had been quickened by mercury, morphia, an acid, or any other retentive of uric acid, precisely similar results would have been produced. Whereas, if the dose of salicylate had been large (15 to 20 gr.) in place of small, the C.R. would have slowed instead of quickening, the granules would have increased instead of diminishing, and the urine would have diminished instead of increasing.

In another figure, obtained in this research, but omitted to save space, a quickening of C.R. similar to that in fig. 2 was produced by 5 gr. of sodium salicylate taken at 7.45 a.m., and came to an end at 10.30 a.m., when a further dose of $2\frac{1}{2}$ gr. produced another quickening, and here the urine increased from 74 c.c. in the slow period up to 210 c.c. in the period of second quickening. And in still another figure the granules were examined every fifteen minutes after a dose of $2\frac{1}{2}$ gr. of sodium salicylate, when they were 1 to 10 before the dose, and in the succeeding periods 1 to 17, 1 to 20, and 1 to 28; the last corresponding with the period of greatest quickening of C.R., and with a fall of blood-pressure from 140 mm., before the dose, to 110 mm. at the time of greatest quickening; this returning again to 140 mm. at 10 a.m., when the C.R. curve had also returned to its original level.

We have here, then, practically complete evidence that the effect of a small dose of salicylate is to clear the blood of uric acid, and to bring about all the effects of this clearance which other retentives can produce—viz., quickening of the capillary reflux, fall of blood-pressure, and a more or less marked diuresis corresponding with the amount of water available for this purpose in the body.

It is interesting to note in fig. 3 that when the salicylate failed to clear the blood of uric acid (being no doubt overpowered by the large amount of alkali present in the early morning) it also failed to produce any of the results which usually follow the clearing of uric acid out of the blood, for the C.R. remained slow, granules numerous, and blood-pressure high. And this leads me to remark that it appears that, in order that retention may be produced with all the phenomena secondary to it, there must be more or less a balance of solvents and retentives. If alkali is present in force it can act as a solvent in spite of a small dose

of salicylate, or if salicylate is in force it can act as a solvent in spite of some alkali; but at a certain point the alkali is just enough to interfere with the salicylate, and the latter is just enough to interfere with the alkali, and then there is no solution at all but retention and its effects, as we see in these figures (compare results of the balance of salicylates and alkali when passed through the uric acid filter, "*Uric Acid*," edition 7, pp. 173, 174). The explanation of these results, as previously given by me in reference to the action of salicylates in arthritis (previous reference), is, I believe, as follows: In the alkaline tide at 9 a.m. the uric acid was in solution with alkali in the blood in considerable quantities, hence the granules were numerous, the C.R. slow, and the blood-pressure somewhat raised; on this there supervenes the absorption of a small quantity of salicylate, which was in the blood in sufficient quantity to prevent solution of the uric acid with the alkali, and yet was not in sufficient quantity to get the uric acid into solution itself. Hence the uric acid was not in solution with alkali, nor in solution with salicylate; in fact, it was not in solution at all, but was more or less completely cleared out of the blood, as demonstrated by the fall in the number of granules. The quickening of C.R., the fall of blood-pressure, the diuresis and the fall of granules are constant results of clearing the blood of uric acid by whatever means this may be accomplished.

While the effect is passing off, the C.R. slowing and the blood-pressure rising again (as from 10 a.m. to 12.30 p.m. in fig. 2), those whose hearts are weak (not necessarily any valvular disease, but weak in muscle tone) may experience a certain amount of fluttering or palpitation, showing that the heart is inconvenienced by the increasing peripheral resistance. The same thing occurs after the exhibition of a nitrite, and I have suggested in "*Uric Acid*," p. 361, that this faltering of the heart before rising blood-pressure after a nitrite may be looked upon as an indication that chloroform may be dangerous for that patient.

The retention produced by a salicylate rises slowly to its maximum in about an hour, and then slowly passes off again; and this slow action and long duration of its effect is a distinct advantage on the side of salicylates as compared with nitrites.

If the above experiments had been performed on a patient who was subject to arthritis, we should possibly have got not only a quickened C.R., a fall of blood-pressure and a diuresis, but also more or less marked signs of pain and inflammation in some of the joints, thus showing where the uric acid had gone. But in the case before us the uric acid was,

most probably, driven out of the circulation into the liver, where it remained without apparently giving any signs locally, and was as gradually returned into the blood in about its original quantity, slowing the C.R. to its original rate in about sixty to ninety minutes, so that the whole fluctuation produced by 5 gr. of salicylate lasted from two to two and a half hours.

If the dose of salicylate had been larger, 15 gr. to 20 gr. instead of 5 gr., there would have been little or no quickening of C.R., no diminution of granules, and no diuresis. The salicylate would itself have acted as a solvent, so that the granules would rather have increased, the urine remained scanty, and the blood-pressure as high as at 9 a.m. (This is an experiment already given in "Uric Acid," p. 95.)

My object in this paper is to point out that the salicylates, which, under certain conditions and in certain doses, are powerful solvents of uric acid and aid its elimination from the body, thus curing troubles due to its local accumulation, are, under other conditions and in smaller doses, retentives of uric acid, hindering its elimination and doing harm in arthritis and other conditions due to local accumulation, while doing good in collæmic conditions associated with excess of uric acid in the blood; that the capillary circulation in either condition is a reliable guide to the results the salicylates are producing; that, when salicylates fail to do good in arthritis or even do harm, it is because something has interfered with their action as solvents, thus preventing their slowing the C.R., increasing the granules in the blood and the excretion of uric acid in the urine; and that, once the cause of this failure has been found out, the result can be controlled—*e.g.*, excess of heat can be met with cold, and excess of alkali with acids.

On the other hand, when salicylates, which have been regarded as solvents, cure a collæmic disease (*e.g.*, headache), it may be because they have really acted as retentives. It has, I think, long been known that salicylates in small doses will sometimes cure a migraine, and I believe that investigation will show that in such cases they have quickened the C.R., cleared the granules out of the blood, and caused a diuresis (as shown in some of the figures), and that the relief or cure of the headache has been the result of this action—*i.e.*, is due to retention and not to elimination of uric acid.

And this has an interesting bearing on some cases one meets with in practice. Thus not long ago a doctor in practice in Chicago wrote to me to ask why his headaches had been worse since he had been taking salicylates, and on investigation it came out that he had been taking

small doses, much too small to produce elimination, and which had, therefore, probably relieved temporarily by causing retention; but as the uric acid was not being cleared out, but, on the contrary, retained, it follows that the headaches would get more and more severe on this treatment, the more the uric acid accumulated. And this is true not only of this particular instance, but of all cases. There is no way out of collæmic troubles by retention, for this must eventually make matters worse, as the quantities of uric acid increase. Much more is this the case when retention is produced by the administration of a xanthin compound (*e.g.*, caffein), for then present relief is due to retention, but the quantity available for future mischief is being increased both by retention and introduction. In the case above-mentioned the headaches improved when the small doses of salicylate were discontinued.

There is evidence then that small doses of salicylate can be used in place of acids, or salts which act as acids, or of morphia and many metals, all of which hinder the solubility of uric acid and so affect the blood and circulation, and which have for this reason commonly been resorted to for the relief of migraine in the past; and all of them, when they produce this result, act like a small dose of salicylate, the action of which has been described in this paper—*i.e.*, clearing the blood of granules, quickening the C.R., and causing a diuresis.

There is also a point in which the retention produced by salicylates has an advantage over that produced by other drugs (*e.g.*, opium), for as opium acts as a retentive chiefly by diminishing peristalsis, and so (by increasing the absorption of acids from the large intestine) raising the acidity, it follows that everything that lowers acidity, such as debility, hot weather, or, still more, diarrhoea, will greatly interfere with the action of opium. Opium will have to overcome the diarrhoea, or raise the acidity in spite of that or general debility and malnutrition, before it can have any effect on C.R. and blood-pressure; but salicylates will not be hindered by these things, as the more alkaline the blood the better will they act as retentives. Similarly, a hot climate is rather against the action of opium, as heat tends to increase the alkalinity of the blood. Hence, no doubt, the natives of India, who depend on opium for their powers of endurance, have to take a larger dose than would be required to produce the same result in a cool climate. A hot climate, as we have seen ("Uric Acid," p. 762), also interferes with the solvent action of a salicylate and favours its action as a retentive.

It may be thought that, in these conditions of high alkalinity, mercury or other metal, which forms an insoluble compound with uric acid,

would act directly and cause retention ; but, as a matter of fact, if mercury causes intestinal irritation or increases diarrhoea, it thus brings so much more alkali into action that its power as a retentive is very greatly diminished. Hence we see that the retentive action of salicylates is so much the more useful because they will often act in debility, malnutrition, diarrhoea or great heat—the very conditions in which other retentives are most likely to fail. But salicylates themselves also will fail if the circulation is so much upset that there is no absorption from the stomach, just as we know may occur in certain conditions of headache or epilepsy (see “Uric Acid,” chapters vii and viii).

When in hot weather salicylates are found to fail in curing acute rheumatism, it will be found that C.R. is quick—*i.e.*, the salicylates are acting as retentives and not as solvents. On the other hand, when in cold weather they are relieving acute rheumatism and bringing down the temperature, the C.R. will be slow and the blood full of granules. It is often quite unnecessary to examine the blood-granules or the blood-pressure, or to measure the urinary water, as the C.R. is the index of it all. The C.R. in the above figures was taken with my capillary dynamometer and a metronome beating half-seconds, but for rough clinical work the point of the finger and counting time will suffice.

A demonstration of the retentive powers of salicylates may sometimes be obtained by taking a small dose (as in one of the figures above described) when exposed to heat and fatigue, and this will also demonstrate the power of uric acid in obstructing the circulation and producing these fatigue conditions. As I have already pointed out (“Uric Acid,” p. 336), there is similar absence of fatigue for many hours on the day after a course of salicylates is left off, there being but little uric acid in the urine and few granules in the blood. This is no doubt partly due to retention at the dead point between salicylates and alkali, as shown in the figures, for it is followed next day by a plus excretion of uric acid (rebound), often accompanied by headache, excessive fatigue, and other signs of collæmia. On the other hand, a large dose of salicylate completely does away with the evil effects of cold, as its solvent action neutralizes the precipitant power of cold, while cold aids the solvent effect of the drug.

And the rate of the capillary reflux will demonstrate, from moment to moment, whether a given dose is, under given conditions, acting as a solvent or as a precipitant ; in the former case C.R. will be slow, in the latter it will be quick. So if salicylates are being given in full doses for the relief of a precipitation disease (arthritis), and if, when so given in a hot climate or hot weather, they neither slow the C.R. nor relieve the

arthritis, we know why they fail, and that it is necessary either to make the conditions favourable for their solvent action (*i.e.*, cool the patient and apply cold to the joints), or to give up salicylates in favour of alkalies whose solvent action is helped rather than hindered by heat, as has been already pointed out in the parallel case of bronchitis—a precipitation disease (see *British Medical Journal*, previous reference).

Lastly, every drug that has any influence on the solubility of uric acid can have its solvent or retentive action demonstrated by exactly the same methods as have been here used to show the retentive action of small doses of salicylates and their effect on C.R., blood-pressure, and the granules in the blood; and C.R. may often be taken as the guide to the results produced, when instruments are not at hand or there is no time for a more complete investigation.

DISCUSSION.

Dr. A. P. LUFF said he would not detain the Section more than a few minutes in discussing that paper, for the simple reason that he considered the matter had been thoroughly discussed often before. Dr. Haig and he were so hopelessly opposed to each other on the question of uric acid as a causative factor of disease that he was sure the author would be willing to believe that he did not speak in any offensive manner towards him when he stated that the paper and its deductions were, in his opinion, based upon a series of fallacies. On the printed slip which had been sent round were the words: "The uric acid controls the capillary circulation, which is quick when there is little uric acid, and slow when there is much in the blood." He asked whether Dr. Haig had ever once estimated directly the amount of uric acid in the blood when it was either little or great. He had not read the last edition of Dr. Haig's book, but he had been most carefully through the previous editions, and although there were constant statements as to the amount of uric acid in the blood, varying under different conditions, not once did he find any estimation of the uric acid in the blood, nor any account of extraction of uric acid from the blood, or the extraction of anything which could be identified by the only reliable test, namely, the murexide test. He could not accept the recognition of the counting of the granules as an index that uric acid was in the blood; he thought it had been proved that those granules had not any relationship to the uric acid in the blood. Again, with regard to Dr. Haig's statement which he had made that evening, and which appeared many times in his book, that the alkalinity of the blood varied: as far as he (Dr. Luff) could gather from the statements in his book, the variations in the alkalinity of the blood were deduced solely from variations in the acidity of the urine. Not once did he find that Dr. Haig had used

any method of directly determining the alkalinity of the blood, such as the simple process of Sir Almroth Wright. Determinations of variations in the acidity of the urine were no guide to variations in the alkalinity of the blood, and it was useless to talk of the blood varying in alkalinity when those statements were solely based upon determinations of the acidity of the urine. It all came back to the question, Is uric acid a poison? He (Dr. Luff) emphatically denied that it was. What experimental proof was there that it was a poison? Large doses had been taken by human beings, and administered to animals, but no toxic effects had followed; there had been nothing more than a little nausea and a transient headache. In leukæmia it was easy to demonstrate a large amount of uric acid in the blood, in a soluble form, but where were the indications that it was toxic? There were none, and therefore he maintained that uric acid was not a poison, and he did not know any diseases or disorders in metabolism which could be directly attributed to it.

Dr. H. C. CAMERON said he was acquainted with Dr. Haig's views, and with the arguments which had been used in opposition to those views; but what he had been particularly interested in was the use of the capillary dynamometer as a means of estimating the uric acid in the blood. Dr. Haig showed a chart with a base-line which was to be taken as a guide for the normal capillary reflux time. He (Dr. Cameron) thought it must be an extremely difficult thing to find such a normal base-line, because, apart from the question whether the capillary circulation was or was not influenced by the amount of uric acid in the blood, there were so many varying factors which did undoubtedly exercise a great influence upon it, such as posture, mental emotion, temperature, or the condition of the digestive organs. It would surely be difficult to place every patient under strictly comparable conditions in relation to all those varied factors, and yet that was necessary to achieve what Dr. Haig had called "the base-line of capillary reflux."

Dr. HAIG, in reply, said the determination of the capillary reflux was an easy thing to carry out, and it could be carried out any number of times a day. As a rule it was done on patients who were in bed, on the front of the chest, while they were horizontal. There the temperature of the skin was that of the whole body of the individual. Among his own cases he had measured the reflux steadily for ten years, and he had curves of his own condition extending back as long. What he had shown on the screen were half-hourly curves, while his own had been morning and evening ones. It was always slower in the morning, and quicker in the evening. In the case of fever it was very quick. In out-patients it enabled one to say whether there was fever or whether it was probable that an individual had Bright's disease. It was a rough guide, and other things must be attended to; but where it was quick the temperature should be taken, as fever cleared the blood of uric acid. Dr. Luff's attitude on the question was not new to him. Several times when he had read papers Dr. Luff had made similar remarks; and he could only say those remarks had not deterred him from pursuing his investigations,

and he did not intend that they should now. It was impossible for the substance referred to in the blood to be anything else than uric acid or xanthin. Anyone who would do the granule test, and do it at intervals for two or three hours, would see that it was impossible for the substance to be anything but uric acid; it came out in the urine as such. He could influence the substance in the blood by half a dozen different drugs. If he had in front of him a patient with a normal capillary reflux, he knew the number of granules he would find. If he then gave salicylate, in one hour the granules would be doubled or trebled in number. If he had a rheumatic fever patient under treatment by salicylate for a day or one and a half days, the granules would be 1 to 1, in place of 1 to 70 before the drug. Similar changes could be produced with every drug in the pharmacopoeia which affected the solubility of uric acid. If the substance was not uric acid, would anyone suggest what it was? Dr. Luff said it was not uric acid, but he did not say what it was. He showed the granules (Mr. Barker Smith's test) before a meeting of the Medico-Chirurgical Society, but the blood-specimen would not keep. Excess of granules was the sign of what he called collæmia. He would undertake to make them in the proportion of 1 to every 1 red cell, instead of 1 to 70 in anyone. He was certain the statement that uric acid was not a poison was not true; he had himself been nearly killed by it several times, and at least one other person in the room had had his life endangered by it, and was saved by measures which affected the uric acid, and which affected that alone. Would Dr. Luff be willing to let him administer uric acid? If so, he recommended him to make his will first. His reply to Dr. Luff's statement that it had been administered without ill effects was that he was aware of this. Sir Alfred Garrod gave some 30 grains, and there was no increased excretion. Why? He himself had injected quantities into monkeys and killed the animals after it, yet there was not a grain of it in the blood. This was because it had gone into the liver and spleen, and there it was found. If a solvent were given, the result would be different. He believed people could be killed with it, but attention must be paid to its solution in the blood, and it must not be merely given as Sir A. Garrod gave it. The number of granules or the capillary circulation would tell when it was in solution in the blood. He could only regard that statement of Dr. Luff's as a joke.

Note on Nutmeg-Poisoning.

By H. H. DALE, B.C.

ABOUT a year ago Professor Cushny¹ made a communication to this Section of the Royal Society of Medicine on Nutmeg-Poisoning, basing his observations, in so far as they dealt with experimental results, chiefly on a paper by his former assistant, Dr. G. B. Wallace. I had, at that time, for some months been making experiments on the action of nutmeg, in connexion with a very exhaustive chemical investigation carried out by Power and Solway.² Until Professor Cushny's communication was published I had not had access to the details of Wallace's results, though I had gathered, in conversation with Professor Cushny, that they were in many respects similar to those which I had obtained. There are some points of difference, however, which seem sufficiently important to mention.

My experiments with the numerous preparations placed at my disposal by Dr. Power had led me to the conclusion, identical with that of Wallace, that the whole activity resides in the volatile oil, and in particular in the substance of high boiling-point which, since Wallace's paper was published, has been chemically characterized and named "myristicin." I agree with Wallace, again, in finding the cat the most sensitive to nutmeg of the animals usually available for experiment. But whereas Cushny concludes, from Wallace's experiments, that "animals correspond very closely to man in their reaction to nutmeg-poison," the effects which I obtained, in my earlier experiments on the cat, appeared to me remarkably inconsistent with the numerous published accounts of nutmeg-poisoning in man. The characteristic result in man of ingesting the substance of one or more nutmegs—that is to say, 5 grm. or more of the drug—would appear from the recorded cases to be narcosis, varied by excitement and delirium, commencing a few hours after ingestion, and usually followed by recovery after twenty-four hours or more. It is stated that death has occurred, but Cushny points out that only one clear case is on record, the case being that of a

¹ *Proc. Roy. Soc. Med.*, 1908, i, (Therap. Sect.) p. 39.

² *Journ. Chem. Soc.*, 1907, xci, p. 2037; *Amer. Journ. of Pharm.*, 1908, p. 563.

child who took a large dose of the drug. The general impression given by most of the clinical records is of a temporary intoxication, which usually passes off without leaving any obvious bad result. One observer even recommends small doses of nutmeg as a mild and safe hypnotic for children. On the other hand, I found, early in the course of my experiments, that the smallest dose of nutmeg, which would produce in a cat any recognizable effect, invariably produced death; further, that the type of poisoning produced was widely different from what I had been led to expect by the clinical records. The following notes on experiments illustrate the nature of my results. It will be noticed that the doses are relatively large, 5 gm. being sufficient to produce marked symptoms in a human being, weighing on an average about twenty times as much as a cat. Smaller doses, however, did not affect the cat perceptibly.

A cat, weighing 2,640 gm., was given 5 gm. of grated nutmeg by the mouth. A small amount was vomited during the night, but no other effect was observed on the day of administration or the day following. On the third day, however, the animal was observed to be jaundiced and very drowsy. It passed into gradually deepening coma, and died about forty-eight hours after ingesting the nutmeg. In a second case, a dose of 10 gm. produced, in a cat of 3 kgm., no effect, except salivation and disinclination for food, upon the day of administration or the two following days. During the next night, however, it passed into very deep coma, and was found in this condition on the third morning after ingestion. The pupils were maximal, the eyeballs quite cold to the touch, there was no trace of corneal reflex. The only signs of life were a feeble, slow heart-beat, infrequent, long-drawn respirations, and the reflex movements which were readily elicited by tickling the pads of the hind feet. The rectal temperature was 26° C. (79° F.). The animal died during the morning—about seventy-two hours from the time of administration. In another case in which 5 gm. were given, nothing was noticed till the fourth day after administration, when fatal coma again appeared.

In all these cases the post-mortem examination revealed the same condition. There was advanced fatty degeneration of the liver, which, in sections appropriately stained, could be seen to affect practically every cell, extending to the middle of the lobules. Local necrotic changes were also obvious. Macroscopically other organs were little affected, but complete microscopical details are not yet available. There was marked jaundice of all the tissues, and the urine drawn off from the bladder

was dark coloured with bile pigment, and gave a cloud of albuminous coagulum on boiling.

The contrast, then, seemed obvious enough. In the cat, a uniformly fatal coma appearing after a few days, always associated with, and probably secondary to degenerative liver changes, not at all unlike those seen in phosphorus poisoning: in man, on the other hand, as the result of relatively much smaller doses, a temporary condition of excitement, followed by narcosis, appearing a few hours after ingestion, and generally passing off without leaving any serious after-effects.

With chemically pure myristicin, the active constituent of the volatile oil, the results obtained were very similar, with the exception that the dose given must be proportionately larger. The difference may with great probability be attributed to the readier absorption of myristicin when it is associated with the fats and other constituents of the whole drug. Given pure it is probably lost to a considerable extent in the faeces, or, if given hypodermically, very slowly absorbed. However, with 1 c.c. of myristicin given by the mouth, I obtained effects closely similar to those which I observed with 5 to 10 grm. of nutmeg. Again no symptoms were observed, except the salivation and defect of appetite, until two or more days after the myristicin had been given. Then the characteristic jaundice appeared, followed by coma, and the post-mortem findings were as before.

So far my results with cats had no point in common with those recorded for man. They resembled the effects obtained by Jürss,¹ who, by injecting relatively very large doses of myristicin into rabbits and guinea-pigs, produced degenerative changes in the liver which he compared to those of phosphorus poisoning, and in some cases recorded a condition of narcosis or coma. I was even in doubt whether the effects in man could be attributed to myristicin until Professor Cushny made his communication, and it became clear that Wallace had produced the primary effects on the nervous system in cats by using even larger doses than those which I had employed. I proceeded, therefore, to try larger doses, and obtained the expected results; 1.5 c.c. of myristicin given by the mouth to a cat of 3 kgm. caused, after an hour, a condition of excitement, with dilated pupils, tremors, unsteady gait and imperfect avoidance of obstacles. The condition was reminiscent of that produced by a small dose of *cannabis indica*, and passed off in a few hours without having deepened into actual narcosis. On the following day the cat

¹ *Schimmler's Bericht*, Leipzig, 1904, p. 159.

seemed rather apathetic, but otherwise normal; late in the day, however, the sclerotics showed signs of slight jaundice. On the third morning the jaundice was marked, and fatal coma followed during the day. In another cat 5 c.c. of myristicin produced excitement and inco-ordination in about half an hour. Complete narcosis followed, lasted for about three hours, and then passed off, leaving the animal practically normal. Again, after an interval of a day, the second stage of jaundice and coma followed. In both cases the post-mortem examination revealed the usual fatty degeneration of the liver.

The meaning of these results seems clear. The primary effects on the nervous system, when produced in the cat, are, indeed, closely similar to those observed in man. But, whereas man is so sensitive to this primary action of myristicin that he can be temporarily narcotised by doses of nutmeg too small to leave any permanent bad effects, the nervous system of the cat is relatively so little responsive that doses considerably in excess of that which will certainly kill the cat are necessary if the primary effects, as seen in man, are to be reproduced. The death of the cat is, in any case, due to secondary coma. This was also mentioned by Professor Cushny, who suggested that it might be attributable to irritation of the stomach and kidneys. My chief point of dissent from his conclusions is that I regard the coma as due to the pronounced degenerative changes in the liver which Wallace seems to have looked for without observing them. The secondary coma I regard as an entirely different phenomenon from the primary action on the nervous system, the two being as distinct, in fact, as primary chloroform anæsthesia and coma due to delayed chloroform-poisoning.

The other point which seems to me to need further emphasis is the very great difference in dosage between man and the lower animals. Whereas one nutmeg, weighing on an average about 5 gm., is said to have produced a pronounced effect in man, the smallest dose with which I elicited the primary effects on the brain of the cat was 1.5 c.c. of myristicin, representing at least 75 gm. of nutmeg. Even if we allow, as apparently we must, that some of the myristicin is not absorbed, we are yet left with a dose which is enormously large in comparison with that which causes very definite effects in man. The fact that man is thus readily affected by doses too small to produce remote toxic effects on the liver seems to me to make the therapeutic application of nutmeg at least worth consideration. The results in the cat, which is unaffected by doses not ultimately fatal, do not afford any strong argument against the recommendation of those who have found in

nutmeg a substitute for chloral, though a more likely use would seem to be as an alternative to cannabis indica.

At the same time the production of secondary coma in the cat must be regarded as an index of a possible danger in the use of the drug except in very moderate doses, a danger which one might, perhaps, on the analogy of secondary anæsthetic poisoning, regard as particularly important in the case of children.

DISCUSSION.

Professor CUSHNY said he thought that Dr. Dale's paper really closed the question of nutmeg-poisoning. When his own paper was read some time ago, Dr. Power suggested that nutmeg might contain some other poisonous principle besides myristicin, but he thought that idea had been disproved by Dr. Dale's paper. His co-worker, Dr. Wallace, did not try to find out whether more nutmeg was needed to poison the cat than man, because it was generally recognized that the nerve poisons acted more readily and in smaller doses in man than even in the cat. In regard to the fatty changes in the liver, he did not know how he had missed them. He would not suggest that the American cat differed in its reactions from the English variety. Their attention was specially drawn to the liver, because, just previously, Lindemann had written about the fat-producing effects of pulegon. The fat was very evident in the liver now exhibited by Dr. Dale, and he did not know how it was missed in his cats; it must have been an error of observation. The fatty degeneration explained the late deaths which Wallace had observed.

Dr. J. GRAY DUNCANSON regretted that—from his standpoint—the most practical feature had not been referred to by Dr. Dale. People in this country did not take nutmeg to cause death, but to produce abortion; and he would like to hear whether Dr. Dale had experimented with it on the lower animals when they were pregnant. Nutmeg was somewhat frequently taken by women, especially in the neighbourhood of London, to bring on a miscarriage, but as it was done surreptitiously it was very difficult for the medical man to ascertain in what form and what quantity it was taken. Those cases which he had seen had been marked by gastro-intestinal disturbance, and it was difficult to know whether to attribute that to the nutmeg entirely, or in part to the gin with which it was most frequently administered. In one case which he saw some years ago there were certainly cerebral symptoms—hallucinations and delusions. It would be very interesting if it could be known, on experimental data, whether nutmeg or its essential principle (myristicin) were emmenagogues.

Dr. DALE, in reply, said he had not experimented with the view of ascertaining whether nutmeg was an emmenagogue, though the point was mentioned by Professor Cushny in his paper some time ago. The purpose of the present communication was to correct the idea about fatty degeneration of the liver. Professor Cushny had just mentioned to him that it seemed to be the fact that several of the drugs causing fatty degeneration of the liver, such as nutmeg, pulegon, and even phosphorus, also had a reputation of being emmenagogues and abortifacients. None of the animals on which he experimented were pregnant.

Therapeutical and Pharmacological Section.

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Counter-irritation.

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THE subject of counter-irritation is a very wide one, and there are many theories with regard to it. It is the practical experience of all of us that it does act beneficially; we know that if we apply a blister to the knee-joint in which there is effusion the effusion may disappear. The same is true with regard to pleurisy. We know also that counter-irritation will relieve pain, and I shall limit my remarks this evening to that aspect of the subject. When we come to inquire into the matter of pain we discover that we know little about the causation of pain and the way counter-irritation relieves pain. But I wish now to bring forward a view which I have been developing for some years with regard to the production of visceral pain. To understand how pain arises we have to understand the tissues in which pain can appear. If you take such a simple stimulus of pain as a pin-prick, or pinching, and apply that to the body, you will find a great variety of reactions, some parts being more sensitive than others. But there is a great extent of tissues and organs sensitive to a pin-prick, and many tissues and organs insensitive to such a stimulus. If this division be made it will be discovered that the organs and tissues which are sensitive to the prick are supplied by cerebrospinal nerves. Those tissues, on the other hand, which show no sensitiveness to a prick of a pin are supplied by sympathetic nerves, and have no cerebrospinal nerves distributed to them. This difference is easy of demonstration, and is accepted by everyone who has inquired into the subject. In pricking any portion of the skin

there is not only pain but also a sense of locality, and I want you to bear that in mind. From the peripheral distribution of the nerves the stimulus passes into the brain, and the brain becomes conscious of a stimulus in a particular locality. If the nerve is stimulated in its trunk, or in the posterior spinal ganglion, as in the case of herpes zoster—which is an inflammatory affection of the ganglion in the posterior spinal root—the pain is not referred to the place stimulated, but to the peripheral distribution of the nerve. If a nerve is irritated in the cord, the pain is not felt in the cord, but at its peripheral termination in the skin. If the stimulus is to the cortex of the brain, the pain is not felt in the brain, but in the skin. Any stimulation of a sensory nerve from its periphery to its termination in the brain causes pain which is referred to the peripheral distribution of that nerve. In the case of a painful cut on the end of a finger, the pain seems to radiate from the cut up the rest of the finger, the usual explanation offered being that inflammation has extended from the sore place; but there is no inflammation or involvement of the skin beyond the cut. As a matter of fact, the injury sends a strong impulse to the spinal cord, which impulse spreads to and affects neighbouring nerve-cells, causing pain referred to areas away from the injury. The same explanation holds for the radiation of pain in toothache; the cheek in such a case may become very sore and sensitive to the touch, but that is not due to the extension of the trouble from the tooth to the cheek. It is due to the stimulus set up by the pain in the tooth going from the tooth to the fifth nerve-centre, and from there irritating the cheek area. This is illustrated in a personal experience of Professor Sherrington. He applied a mustard plaster to his chest, and he had in a short time pain at both his elbows. The reason is that the cells in the spinal cord of the nerve-supply of the skin of the chest and of the elbows are contiguous, and the violent stimulus from the skin spread in the cord to neighbouring nerve-cells.

I have said the viscera are insensitive to pain when a pin-prick is applied: you can cut, tear, burn, and suture the bowel, the liver, or the spleen without causing any sensation of pain. But pain can be caused by the viscera, as we know. The reason the prick in a viscus does not produce pain is that the stimulus is not adequate; if there is an adequate stimulus, pain will arise. Such an adequate stimulus does arise in renal calculus and gall-stone colic, yet no pain will be caused if you cut the ureters or gall-bladder. It has been said that the viscera are so poorly supplied with nerves that they do not readily convey sensation; but if you take a portion of bowel or ureter and cause spasmodic contraction

of it, there is intense pain. The action I believe to be this: Normally there are nerves passing from the viscera conveying stimuli to the cord which cause no sensation; if the stimulus is adequate it spreads in the spinal cord to the neighbouring cells, and whatever cells it comes across it stimulates to activity. If it is a motor cell it causes muscular contraction; if it is a sensory cell it causes pain; if it be the nerve to a viscus, such as the bladder, there will be irritation of the bladder. In the case of renal colic from renal calculus there may be acute pain in the testicle, simply because the stimulus, when reaching the spinal cord, has irritated the nerve-supply to the tunica vaginalis.

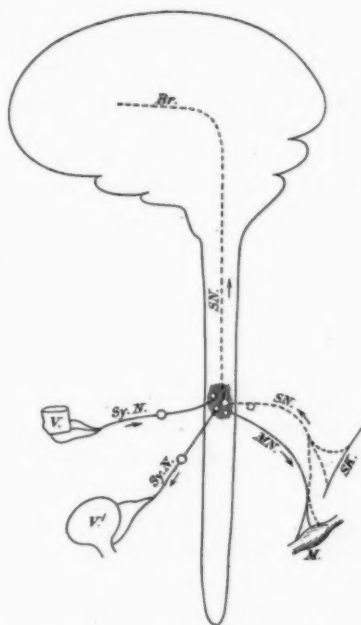


Diagram to show the mechanism of visceral pain and the manner by which it is relieved by counter-irritation. If a stimulus of an adequate strength arises in a viscus (V.) it passes by the sympathetic nerve (Sy. N.) to the spinal cord, and there affects the nerve-cells in close association with the cells of the sympathetic nerves. If the stimulus spreads to a cell of a sensory nerve (S.N.) pain is perceived by the brain (Br.), but the pain is referred to the peripheral distribution of the sensory nerve in the (Sk.). The sensory nerve can thus be affected from two sources—visceral (V.) and cutaneous (Sk.). If a blister be applied to the skin (Sk.) where the pain is felt, the irritation from the blister takes possession of the sensory nerve and excludes the stimulus from the viscus (V.).

I propose to limit the application of my remarks to a case of gastric ulcer. I have observed during many years an enormous number of cases, and kept records of them. In 95 per cent. of cases of gastric ulcer, where there is a boring pain limited in extent, it will be felt in the epigastrium. If the ulcer is at the cardiac end the pain will be felt at the upper part of the epigastrium ; if the pain is at the lower part of the epigastrium the ulcer will be situated at the pyloric end of the stomach. I have had many opportunities of verifying these statements by post-mortem examinations and at operations. At the area of pain there may be hyperalgesia of the skin and deeper tissues and a contraction of the rectus abdominis. I show you a diagram of a case of gastric ulcer which I had under observation ten years. We used to treat her with blistering, and when she died the mark of the blister was present in the skin of the epigastrium. When the post-mortem examination was made my colleague made a careful note of the site of the ulcer. He found it in the pylorus, well away from the region of tenderness shown by the mark of the blister. Of course, it may be said that may have been a post-mortem change, and the conditions may have altered. Many believe the ulcer is immediately beneath the tender area ; and one eminent physician said if he were to pass a needle through the seat of pain he would penetrate the ulcer ; but when the patient breathed the stomach and the ulcer moved up and down, while the pain remained stationary. How is the pain produced ? I suggest the stimulus arises in the ulcer, passes to the spinal cord, and is of such intensity as to irritate the sensory nerves, and the pain is felt in the epigastrium, according to the law that stimulation of a sensory nerve causes a pain referred to its peripheral distribution. If the irritation from the ulcer be continuous, then it produces an irritable focus in the spinal cord, as shown by the hyperalgesia of the skin and muscles in the epigastrium and by the contraction of the rectus muscle. In the patient referred to there was intense boring pain in the lower part of the epigastrium for many hours every day. We put on a blister on the painful spot, which was as large as half-a-crown. When the surface was raw she was free from pain ; when the part healed up again she was as bad as ever. This was repeated many times until savin ointment was used to keep the surface raw ; while the surface was raw the patient experienced great relief. The explanation I suggest is that the sensory nerves passing from the epigastrium to the brain were stimulated in the spinal cord by the stimulus from the gastric ulcer ; but when a blister was applied to the epigastrium, or when the surface was kept raw by the savin

ointment, the stimulus from the raw skin was sufficiently powerful to exclude the stimulus from the gastric ulcer. This explanation would be in accordance with Professor Sherrington's demonstration—that certain nerves are the common paths to stimuli from various sources, and that when the stimulus from one source is sufficiently powerful it excludes the stimuli coming from the other sources. I take this simple illustration as we recognize the original cause of the pain from the gastric ulcer, a limited area in which the pain was felt, definite relief obtained by counter-irritation, and sufficient data for which a reasonable explanation could be given.

DISCUSSION.

The PRESIDENT (Dr. Burton Brown), after thanking Dr. Mackenzie for his interesting opening, said the subject reminded him of the French criminal who was condemned to be broken on the wheel, and who laughed at the second blow. When asked why he laughed, he said he thought he would suffer as much from the second pain as from the first, but instead he felt very little; the first so exhausted his pain that he had none left for the second blow. Soldiers and sailors who were condemned to be flogged were in the habit of biting hard on a leaden bullet, as it was found that exertion seemed to diminish, but not to obviate, all pain.

Dr. W. ARMSTRONG (Buxton) said that although the opener's remarks had somewhat circumscribed the subject, he (Dr. Armstrong) assumed that the general subject of counter-irritation was under debate. He had found good results in gastric ulcer pain from counter-irritation over the splanchnics; the results seemed to be better and more lasting than when it was applied over the pit of the stomach. The use of the long-neglected seton had given excellent results in obstinate cases of migraine. He had some thirty cases in which it had done much good, though in these fastidious days people were reluctant to submit to that treatment. He had long been using the electric cautery with excellent results in many refractory ailments connected with the sympathetic nervous system. Many cases of neurasthenia, some in which there were even delusions, and of the worst forms of insomnia, had given way in a remarkable manner to the application of the electric cautery over the cervical ganglia. His best results from spinal counter-irritation had been in cases of rheumatoid arthritis, following Professor Latham and Dr. Midelton, who had got good results from applying large blisters over the cervical enlargement of the cord for joint-affections of the upper extremity, and over the lumbar enlargement for those of the lower. He had steadily carried out that line of treatment for the last five years, of course always trying to find out and deal with reflex sources of irritation. The results were better than from any other single method of treatment. He regarded the electric cautery as much the best agent

for counter-irritation ; neither a raw surface nor discharge were necessary, and it could be persisted in for weeks at a time, the pain being very slight.

Professor CUSHNY said it was a great joy to one who attempted to show that the old masters were good observers—better observers than their descendants—to find that one of their methods was being explained. Forty years ago the average therapist was liable to incur considerable ridicule if he attempted to defend counter-irritation ; but the recent work of Mackenzie, Head, and others had shown that there was a great prospect of being able to explain, at all events, the results. Dr. Mackenzie had chiefly brought out the subject of referred pain in the skin. Since looking into that gentleman's work recently, he had concluded that a certain amount of the benefit of counter-irritation might be due to the influence on deeper structures. A certain part of the pain in the viscus was referred pain from the skin ; but some of it arose not from the skin, but from the muscle underneath contracting. In lumbago and a certain number of so-called rheumatic conditions the pain was largely due to a contraction of muscle ; a roll of stiff muscle in the neck or in the lumbar region could often be made out. If a counter-irritant were applied over a muscle where it was painful, not only did it influence the skin circulation, but, as had been shown experimentally, the circulation of the muscle was improved. Lazarus Barlow showed, years ago, that the whole body-wall, for a considerable depth, was better supplied with blood when a blister was applied to the skin. So that a part of the explanation might be due to the better supply to the muscle and its consequent relaxation. Dr. Mackenzie's explanation, by which the relief of one pain was due to the setting up of another, was to him still a little obscure ; still, Mackenzie's work had afforded some foundation for the belief in the use of counter-irritants. Of course, by applying a blister and causing pain one might not only act upon the pain, but the viscus might also be considerably influenced, the action being a reflex one.

Dr. SOPER thought the subject demanded great consideration, and it was a good sign of the times that practitioners were beginning to appreciate the efficacy of counter-irritation. Fifty years ago he saw very much more heroic treatment than anyone present would dream of applying. At that date it was not uncommon to apply the actual cautery to the skin for pain ; or to make an issue with *potassa fusa*, dressing it every day with resin ointment ; and applying a blister measuring 8 in. by 4 in. in the case of pneumonia was quite common. The effect upon a child was marvellous. But so different was the present-day physique of persons that a cantharides plaster would be approached with fear and trembling. When the apprentice had a much freer hand, in the old days, he had seen marvellous results from blistering and bleeding. He had noticed that the Londoner stood pain much worse than the country dweller. He strongly endorsed what had been said about the seton and its action on the deeper structures. In a case now under his care he would probably go down $\frac{3}{4}$ in. and leave the seton in for six weeks, and if it did not discharge freely he would resort to the old plan of dressing with resin ointment.

The Treatment of Gastric Ulcer by Immediate Feeding : based on a comparison of Cases on the Lenhartz Dietary and Cases treated by Saline or Nutrient Enemas and a Graduated Milk Diet.

By EDMUND I. SPRIGGS, M.D.

IN December, 1906, in a discussion at the Medico-Chirurgical Society [14], and in the following year in the *Clinical Journal* [15], I mentioned the favourable results which had followed the use of the Lenhartz method of treatment of ulcer of the stomach in a few cases under my care. Since then I have had the opportunity of observing more patients treated in this way, and propose this evening to lay the results before you. The cases here reported were unselected ones of the ordinary types met with in hospital practice. For the purpose of comparison a similar number of other cases taken consecutively from the records of St. George's Hospital during the same period and treated by the usual methods are tabulated with them. Before proceeding to consider the tables in detail, some reference will be made to the principles upon which the Lenhartz diet is based. Secondly, a summary of the routine employed will be given; and, thirdly, the advantages and disadvantages of the method will be discussed and compared with those of the usual plan of treatment. We will then turn to the cases observed, and endeavour, so far as the clinical material serves us, to form an opinion as to whether the claims put forward by Lenhartz and his followers are justified.

The essential features of Professor Lenhartz's treatment are: (1) Complete rest in bed for four weeks; (2) feeding the patient from the beginning of the attack with small quantities of beaten-up egg and milk, the quantities being increased daily; (3) the application of an ice-bag to the epigastrium; (4) adding to the dietary boiled rice, mince, and other semi-solid and solid foods after the first week; (5) the administration of bismuth and iron in suitable form. Of these the complete rest in bed, the bismuth and iron, and the ice-bag are commonly advised by physicians. But the plan of allowing food to be put into the ulcerated stomach at the beginning of treatment is contrary to the usual practice. The method is founded on the view that acid gastric juice delays the

healing of an ulcer, even when no food at all is put into the stomach; and that the reparative process cannot proceed satisfactorily in an ill-nourished and anæmic person, such as the subject of this malady commonly is. Experimental researches are quoted in support of each of these propositions. That the acid juice will irritate an open sore everyone will agree. Mathes [*vide* 18] produced artificial lesions of the gastric mucous membrane in animals, and found that they healed rapidly, except when exposed to the action of dilute hydrochloric acid. This result appears to show that healing was not delayed by the presence of a normal amount of acid, but of an excess. In the subjects of gastric ulcer, however, there is a general agreement that the normal gastric juice, if secreted, will act prejudicially, and the treatment by nutrient enemas is intended to prevent the secretion of juice which normally appears when food is introduced into the stomach. But it must be remembered that gastric juice also flows on the thought of food in a hungry person, and it has been stated by Bourget, Winternitz and Umber that when a nutrient enema is given pain is felt in the stomach. Ewald and Michael, however, were not able to confirm the occurrence of secretion. Pain is certainly felt by many patients, and is often referred to the stomach, though in some cases it may arise in the colon [3]. Obviously, if either the thought of food or the irritation of an ulcer may be followed by the production of juice, harm may be done, for the juice will flow over the raw surface, which is unprotected by the normal mucous membrane.

The second point, namely—that the reparative process cannot proceed so well in an ill-nourished and anæmic person—is self-evident; it is also clear that even if the patient is, as the subjects of gastric ulcer often are, fairly nourished at the beginning of the attack, the treatment by saline injections or nutrient enemas will be followed by wasting, for all who have studied the subject of rectal feeding by means of accurate metabolism experiments are agreed that, although a considerable amount of energy may be furnished in this way, sufficient food to maintain the needs of the body cannot be supplied [16] [17]. Still less is the anæmia from which these patients suffer likely to improve on such a dietary. Quincke and Daettwyler [8B] found that ulcers which healed quickly in normal dogs did not in those rendered anæmic. If food can be introduced into the stomach from the beginning, the recovery from the anæmic condition is likely to be more rapid than if the patient is subjected to a further period of subnutrition. The objection to immediate feeding by the mouth is that work is thereby thrown upon a

diseased organ which, it might be thought on rational principles, should be given a complete rest. Further, it is urged that the introduction of food into the stomach necessitates the contraction of that viscus to pass it on into the duodenum, and that such movement of the stomach walls is likely to dislodge a clot in an opened vessel and cause a recurrence of hæmorrhage. To this Lenhartz [7] replies that the danger of a clot becoming dislodged in this way is no greater than the danger of its being dissolved by some of the gastric juice in the stomach, and that if small quantities of protein food are supplied the hydrochloric acid of any gastric juice poured out is neutralized, because it combines with the protein. To this end small quantities of beaten-up egg and milk are used. Egg albumen rapidly forms a combination with hydrochloric acid. Milk is a food that calls forth less secretory activity in the stomach than any other food. Fat, which is present in the yolk of an egg, is known to inhibit the secretion of juice. So that the particular combination of foods used is that calculated to excite the least possible secretion and to combine most efficiently with the acid of any juice secreted. As the food is given in very small quantities at a time, distension of the stomach by food is obviated and the necessary movements reduced to a minimum. It is probable that distension of the stomach is far more harmful to an ulcer than contraction. The food is swallowed and requires no mastication, and this again keeps the gastric secretion low, for the act of chewing is accompanied by a production of juice. An ice-bag placed upon the epigastrium is intended to keep the stomach in a quiescent state. The application of cold has been observed by Rossbach [11A] to diminish the movements of that organ. By promoting constriction of the walls gaseous distension, which might be a cause of hæmorrhage, would be prevented. An ice-bag also relieves pain when present.

The routine of the Lenhartz method is as follows: The patient is kept absolutely in bed for four weeks, for the first two of which she is not allowed to move from the supine position for any reason whatever. All mental excitement must be avoided. An ice-bag is kept upon the stomach almost continually for the first two weeks. The dietary consists of eggs beaten up with sugar, or, in some cases, with wine, and iced; and of milk. These two foods are taken in small quantities at frequent intervals from a teaspoon, the amount prescribed being spread over the day, and not given at definite meal-times. The first day 7 oz. to 10 oz. of milk are given, and one egg. The quantity is increased daily by 3½ oz. of milk and one egg until 1½ pints of milk and

six eggs, or, in some cases, eight eggs are reached. From about the third to the eighth day raw, or almost raw, mince is added, starting with an ounce, in divided doses, either beaten up with eggs or alone; the next day, if well borne, 2 oz. are given. In these cases minced beef was used.

From the seventh to the eighth day boiled rice is added to the dietary, and then some softened bread, and then a small quantity of bread and butter. The diet is then gradually increased by the addition of more mince or pounded fish until by the end of the fourth week the patient is on an ordinary mixed diet containing the common foodstuffs, with the exception of indigestible solids, such as peas or other seeds. The patient is instructed to masticate very slowly. On the twenty-eighth day she is allowed to get up, and is discharged from the sixth to the tenth week. For the first ten days bismuth subnitrate is given in doses of 30 gr. in water without mucilage twice or three times a day. From the sixth to the tenth day sulphate of iron is given in the form of the following pill: Sulphate of iron, 150 gr.; calcined magnesia, 20 gr.; glycerine, 1 dr. Mix and divide into sixty pills. Two of these are given two or three times a day. Lenhartz increases the dose gradually, giving three for three days, four for four days, up to ten for ten days, and then down again. In some cases arsenic is added. The bowels are not disturbed at all during the first week, unless they are naturally opened. An enema is then given and repeated every fourth day during treatment. The mouth should be washed out regularly and attended to.

In my own cases, I have usually begun with $3\frac{1}{2}$ oz. of milk and one egg on the first day, and I have seldom given the iron pill more than three times a day. The oxychloride or the carbonate of bismuth has been used instead of the subnitrate.

It is claimed for the Lenhartz method that it is suitable for all forms of gastric ulcer, except those associated with mechanical deformities, such as stenosis of the pylorus and those with some serious complication, such as perforation, peritonitis, and subphrenic abscess. It is said that the sour regurgitation, the vomiting, and the pain and distress after food disappear in from a few hours to a few days. The improvement is certainly rapid, and the body weight may increase in the first week. It is said that the method has been found successful after recurrent hæmorrhages have occurred on rectal feeding. It is important that patients should be kept in hospital some weeks after they have reached full diet, for a case cannot be recorded as cured until not only are there no symptoms, but the percentage of hæmoglobin and the weight are

normal. Unfortunately, it is difficult to persuade patients of the hospital class to remain under treatment after they feel well.

This diet introduces an amount of food which soon becomes adequate to the needs of the body. A table is given by Lenhartz in which the calories *per diem* are shown to reach nearly 1,600 on the seventh day, and 2,500 on the tenth. The actual figures for the first fourteen days are: 280, 420, 637, 777, 956, 1,135, 1,588, 1,721, 2,138, 2,478, 2,941, 2,941, 3,007, 3,073.

Before recommending a new method it is necessary to ask whether the old is unsatisfactory. There is no doubt that the recognized method of treatment is, in many cases, extremely successful. It has, however, some serious disadvantages. In the first place, severe cases are subjected to a considerable period of starvation or semi-starvation, and the less successful the treatment is, the longer is the starvation period extended. The routine, when fully carried out, is extremely tedious. Dr. Hawkins [5] recommends, for instance, a course in which, four weeks after admission, the patient is having only milk, plasmon, arrowroot, and thin bread and butter; fish and chicken are to be started six weeks after the mouth-feeding has begun. There has been, however, distinct reaction among physicians against this extended period. During the period of enemas the mouth may become very foul in spite of careful washing out, and parotitis has occurred (*vide* Rolleston and Tebbs) [9]. Vomiting may be very troublesome in some cases, and it is interesting to note that Drs. Rolleston and Jex-Blake [10] found in one case that on cautiously dieting with solid food and withdrawing the enemas it ceased. The prolonged period of subnutrition is accompanied by considerable acidosis, as Rolleston has shown, but stronger than even these objections is the unpleasantness of rectal feeding. In hospital practice this is not such an important point, although the patients and the nurses will take a very different view to that of the physician, who has nothing to do with the actual carrying out of the treatment. In private practice, however, the case is different. Even if the Lenhartz method is not superior to the ordinary method, if it is equally good and not less safe it will prove a boon both to the doctor and the patient when treatment is being carried out at home, where rectal medication is nearly always objected to, and very often inefficiently administered.

In the following tables the details of 33 cases treated by the Lenhartz method and 34 cases treated by the ordinary methods are given. The cases on the Lenhartz treatment which were not under my own care during the whole period of their stay in hospital are marked with

an asterisk. I am indebted to my senior colleagues (Drs. Rolleston, Ogle, and Latham) for permission to publish these. The cases were taken seriatim and not specially chosen, with the exceptions mentioned below. The 34 cases treated by other methods are taken consecutively from the hospital records. The only exceptions that have been made in both tables are those of patients in whom the diagnosis was, in my opinion, doubtful, either because the symptoms were not severe enough to justify the diagnosis of ulcer, or because the illness was thought or proved to be due to carcinoma; and cases severely complicated by other diseases not directly connected with the gastric ulcer. A few cases have also been omitted in which the patients were treated by immediate feeding, but in a manner widely different to that advocated by Lenhartz. Some of these did well and some badly, but it would be difficult to decide in which table to put them. On the whole the two series may be taken as similar in type and in severity.

Both series contain examples of the usual accompaniments of gastric ulcer—namely, anæmia, carious teeth, and neurosis. The Lenhartz series includes one case with albuminuria and one with chronic alcoholism. The second series contains one patient with ulcer of the leg, one three months pregnant, and one with uterine displacement. (See Tables I and II, pp. 94-107.)

In considering the advisability of adopting the treatment by immediate feeding, the first question raised will be: is it dangerous? is there a greater risk of the recurrence of hæmorrhage and perforation than by the usual method of nutrient enemas followed by a graduated milk diet? In the above cases in the first series 25 of the patients had *hæmatemesis* in the attack for which they were admitted, and one had had it in a previous attack. In the 25 cases there was one recurrence on the third day of treatment. The patient was not under my care, and the method was not being rigidly followed, inasmuch as no ice-bag was prescribed and the patient had moved from the recumbent position when the hæmorrhage recurred. The feeding was discontinued, the patient being put upon nutrient enemas, and a good recovery was made. In the 34 cases treated by other methods, 29 suffered from hæmorrhage in the attack for which they were admitted. There were four recurrences, two on the second and two on the third day. In one case there was bleeding on both the second and the third day. As far, then, as these figures go the danger of hæmorrhage is certainly not greater and is apparently less upon the Lenhartz diet.

It may be objected that some of these patients were not suffering

from ulcer, but from the gastrotaxis described by Dr. Hale White [19]. This criticism, however, will apply equally to cases in both tables.

The researches of Dr. Sidney Martin, Dr. C. H. Miller [8A] and others indicate that the pathology of a gastric erosion and of a gastric ulcer is identical, for each appears to arise in a mass of lymphoid tissue. Lymphoid tissue is more abundant in the stomachs of those who are subjects of dyspepsia, and the absorption of irritating material from the stomach may cause a breaking down of the follicle. We know that in the small intestine also the lymphoid patches are the most vulnerable areas. Dr. Hale White has demonstrated that symptoms formerly thought to be definitely indicative of an ulcer may arise from a minute fissure or a group of bleeding spots. These bleeding spots, however, are found frequently in that region of the stomach most prone to ulceration, and it is difficult to say that a group of such small lesions may not become an ulcer.

We may next consider the question of *pain*. On the Lenhartz diet the usual result is for pain to vanish entirely within forty-eight hours of the commencement of the treatment, and the diet is so graduated that the risk of recurrence of pain when solid food begins to be taken is, in my experience, less than that of the ordinary method of treatment, in which it is a common thing to find the patient do well until the attempt is made to take fish or mince.

In the 33 cases treated by the Lenhartz method, in eight cases pain is mentioned in the notes after the seventh day. Three of these were transient recurrences. One was an extremely neurotic patient, who also appears in Table II, and on both methods of treatment complained of pain during most of her stay in hospital, and another was the subject of nephritis. In three others of the 33 cases pain was complained of after the first week.

In the 34 cases treated by other methods, in 11 pain is recorded after the first week. Two of these were neurotic women, one being the above-mentioned case. In two others hæmorrhage had recurred; one was a patient upon whom gastro-enterostomy had been performed; and there were six others about whom no special note need be made except that the pain persisted. In this respect, therefore, the advantage again seems to lie with the Lenhartz treatment.

My experience is in agreement with that of Lenhartz, in that in no case was it necessary to give opium or its derivatives for the relief of pain.

Weight.—Of the patients on the diet who were weighed on entering the hospital, eight put on an average of 4·2 lb. each, whilst six lost an

average of 3.5 lb. The figures are of little value, however, as they do not reckon the increase of weight which took place at the convalescent hospital. Of the patients who lost weight one was the patient who had nephritis (in whom the diet could not be fully carried out) and lost 6 lb. (Case 2), and another was a case (No. 4, Table I, No. 21, Table II) who yielded to neither method of medical treatment nor to operation.

Parotitis is liable to occur in patients being fed by the rectum, even when the greatest care is taken to wash out the mouth with cleansing fluid. It is recorded in two of the 34 cases treated by the other methods, but in none of those upon the Lenhartz diet.

The figures are too small to judge of the *mortality*, but it will be seen that no case died upon the Lenhartz diet.¹ There was one death in the cases in Table II.

The *length of stay in hospital* averages thirty-four days in both sets of cases. In this connexion it must be noted that many patients are sent from St. George's Hospital to the Convalescent Hospital at Wimbledon at a much earlier date than it would be possible to send them to their homes. This figure, therefore, cannot be compared with hospitals not having a convalescent branch of their own near at hand. Cases of gastric ulcer do uniformly well at the Convalescent Hospital, and almost invariably put on several pounds weight. Fourteen of each series were sent to Wimbledon at the end of their stay in the hospital.

Especially instructive is the comparison of the *number of days* in the two sets of cases *before the patient reached a diet containing meat or fish*. In the patients treated on the Lenhartz system, 14 patients were upon the ordinary diet of the hospital, containing meat, fish and potatoes, before leaving, the average time taken to reach this standard being thirty days. Eleven cases left the hospital on a fish diet, which includes potatoes, having reached this diet in an average of twenty days from the commencement of treatment. Six other cases were taking fish, but not the full fish diet, when they left the hospital. In the 34 control cases the result is very different. Only four took the full ordinary diet before leaving the hospital, having reached this stage in an average of twenty-three days. The large majority—namely, 22 patients—did not reach the stage of meat, leaving the hospital on a fish diet, which they were given in an average of twenty-two days after admission.

¹ Since writing the above I have seen a case in which an extensive chronic ulcer, in an elderly woman, perforated whilst upon a modified Lenhartz treatment, with a fatal result. The patient had left St. George's Hospital free from pain, after this treatment, a fortnight before, but relapsed. The perforation was undiagnosed.

Seven others were taking fish, but not the full fish diet. The average period before taking fish in these cases was seventeen days. From a consideration of these figures it is evident that the Lenhartz method of feeding enables the patient to take an ordinary diet without pain in a shorter time than treatment by nutrient enemas and a graduated milk diet.

In two of the cases in the first series, and two in the second, resort was had to *gastro-enterostomy*, which should be performed when medical treatment has repeatedly failed. In one further case in the first series the operation has just been done on my advice in a quiescent interval, as, although the patient responds excellently to medical treatment, she has had several recurrences. A small ulcer was found on the lesser curvature adherent to an indurated area of pancreas.

It is important to bear in mind that the medical treatment of this disease should be carried out with as much care and precision as is required for surgical treatment. If this be done it will be found to be much more efficient than many surgeons appear to think. As I have before pointed out [14], any patient who has been treated on milk or upon nutrient enemas and has not recovered is regarded by some surgeons as a fit subject for operation. Whilst not minimizing the beneficial results of *gastro-enterostomy* in intractable cases, it must be pointed out that this operation is not an infallible cure for gastric ulcer. In the present small series it will be noted that three of the patients reapplied for treatment after this operation had been performed, and I have seen a number of others in the out-patient department and elsewhere with recurrence of symptoms. Another reports that she is still suffering from pain and vomiting. Whether cases of gastric ulcer are operated upon or efficiently treated medically, the majority, for the time being, do extremely well, and the mortality from the disease in nearly all the published series of cases, as I have shown elsewhere [14], is extremely low. *Gastro-enterostomy* is not a dangerous operation under ordinary circumstances, but, as Mr. Paterson has shown, it is a dangerous operation when performed for hæmorrhage. I believe, therefore, that if this operation is to be performed it should be done, firstly, in cases which after prolonged medical treatment do not yield, and, secondly, in cases which, having been repeatedly treated by medical means, recur. Perforation and mechanical deformities of the stomach must, of course, be treated surgically.

It will be seen in the tables that in many cases the diet was not carried through in its entirety. My own impression is that the more thoroughly the treatment was applied the better the results. But I

need hardly add that no physician will treat even the commonest complaints by rule of thumb, or will have any hesitation in modifying the prescribed formula when it seems best to do so. I have found, however, in the patients under my own care that the need of much modification is the exception rather than the rule. To a few patients the diet is objectionable. Some prefer the egg and milk mixed, and some alternate teaspoonfuls of milk and of beaten-up egg with sugar. The mixture of a little wine with the egg is also recommended, but I have not found this necessary. If the beaten-up egg excites nausea to the point of vomiting, beaten-up egg-white without the yolk may be used. Instances are reported, and similar ones have come under my own notice, in which, in spite of a strong dislike to the food in the first few days, excellent progress has been made. As a general rule the patients are comfortable, happy, and appreciative of the relief afforded them.

As regards the subsequent history of the patients, I have been able, through the lady almoner of the hospital, to hear from 21 of the first series and 16 of the second. Of the 21 patients treated by the Lenhartz method who have been traced, 6 have been well since leaving the hospital, 9 have had a return of the symptoms of gastric ulcer, and 3 suffer from indigestion. Of the 16 patients traced who were treated by the usual method, 6 have been well since leaving the hospital, 8 have had a return of the symptoms of gastric ulcer, and 2 suffer from indigestion. The two series are not absolutely comparable in this respect, for ten months longer have elapsed since the last patient in Table II left the hospital than is the case in Table I. As the figures stand, however, we may conclude that the results are at least as durable as those of treatment by nutrient enemata and a graduated milk diet.

A few statistics and criticisms of the Lenhartz method have been published:—

Wagner [18] in 1904 reported 60 cases, in 35 of which there was no pain from the beginning, and in only six did pain last beyond the first week of treatment. In one case the pain continued during the whole stay in hospital.

Haberman [4] in 1906 reported 135 cases treated by Professor Lenhartz in Hamburg. There were three deaths, each of these fatal cases being very severe or complicated. A recurrence of hæmorrhage took place in 8 per cent. as compared with 20 per cent. in 100 cases treated by the old method. Most were dismissed cured before the eighth week, and no unfavourable effects were produced.

Wirsing [20] reported 42 cases in 1906, 14 of which had had recent hæmatemesis. In one case hæmorrhage recurred. In 27 of these patients the acidity of the stomach contents was estimated, and it was found that the amount and percentage of hydrochloric acid was diminished during the treatment, the latter on the average from '14 to '11 per cent. Some cases did not show this and yet progressed as well as the others.

The method of Senator, which has some similar features to that of Lenhartz, may be mentioned here. It consists of the administration of gelatine, butter and cream from the beginning. He [13] has reported 50 cases fed upon this diet, of whom two died, that is 4 per cent.

Ewald [3] uses nutrient enemas for three days after a hæmorrhage and then gives milk, butter, and eggs, followed by other foods as in Lenhartz's plan. He reported 34 cases in 1906. The results, however, appear to be inferior to those of the Lenhartz method. In 14 cases death occurred either after operation or immediately after hæmorrhage, and there was a recurrence of hæmorrhage in 7 per cent.

Ewald considers that the Lenhartz method cannot be justified on theoretical grounds and is not worth the risk. The above facts and figures, however, do not support this view, and so far as data have been collected it appears to be less risky than other methods of treatment.

Lenhartz [7] in 1907 reported 140 of his cases, all with recent hæmorrhage by the mouth, or melæna. The mortality in these was 2'14 per cent. He recommends that in such cases great care should be taken not to increase the bulk of milk too rapidly, and in some of the instances quoted the eggs were given without the milk for two or three days. In one case by the twelfth day only 17 oz. of milk per day was being taken. He lays great stress on the instruction that the stomach should never be distended. He reports that in two of his patients who died on the fifteenth and eighteenth day respectively the ulcer was found to be smooth and healed. He recommends the diet strongly for patients who have recently had a gastro-enterostomy performed.

Lambert [6] in 1907 published five severe cases, all of whom did well on the treatment. One was a woman aged 32, who, after seven days' rectal feeding and seven days' careful feeding by the mouth with peptonized milk, still had occult blood in the stools and a hæmoglobin percentage of 39; the question of operation was mooted, but the Lenhartz treatment resulted in a cure. Another patient objected to the diet even to the point of nausea and vomiting, but, nevertheless, after the cessation of the diet for one day, did well. In another

hæmorrhage went on until the thirteenth day, and the case was regarded as below the safe limit for surgery, and yet made a good recovery on this treatment. A fourth case with signs of peritoneal irritation, leucocytosis, and a temperature of 100° F. to 104° F. also recovered. In a fifth case in which the hæmoglobin was reduced to 20 per cent., in spite of an attack of enteric fever upon the twenty-second day, the treatment proved successful. Lambert's fourth case resembles one which came to my notice after the figures in Table I had been collected. A young woman of neurotic temperament was treated by Mr. G. E. Friend for gastric ulcer on the Lenhartz plan. She had no pain for four days, when vomiting recurred with rigidity of the upper part of the abdomen. Perforation was discussed; the patient was put upon enemas and made a good recovery. It is a matter of conjecture whether she would or would not have recovered equally well if, as in Lambert's case, the treatment had been persisted in.

Berger [1], in a publication from the Hamburg School, points out that so efficient has the method of treatment been found in the Eppendorfer-Krankenhaus that it has proved useful as a method of diagnosis, for in cases which fail to respond the diagnosis has often been at fault. He quotes six cases in which the failure of the Lenhartz method led to the diagnosis of cancer, which was confirmed in four of them by operation, and in two by autopsy. In another case hæmorrhage was found to arise from dilated veins about the œsophagus, due to cirrhosis of the liver. He says, further, that in cases of pain without bleeding which do not yield, nervous disease may be suspected.

Schnütgen [12] mentions, in 1907, that Lenhartz had then treated 201 patients with a mortality of 3 per cent. This is about the same as the mortality in 195 cases treated by nutrient enemas by Leube, which was 4 per cent. Leube has altogether collected 556 cases with a mortality of 2·2 per cent., but many of these were not of the hospital class.

We may conclude, therefore, that the death-rate of patients treated by the Lenhartz diet is not greater than that of those treated in other ways.

Finally, Dr. Langdon Brown [2], in a recent paper, mentions that he has treated 11 cases by the Lenhartz method with good results in nine of them.

In conclusion, from an examination of these two series of cases it may be said, *first*, that the Lenhartz method of treatment is not more

dangerous than treatment by nutrient and saline enemas, followed by a graduated milk diet. In these particular cases the recurrence of hæmorrhage was less frequent, and there were no deaths.

Secondly, that the pain suffered by the patient in the course of treatment is less on the Lenhartz diet.

Thirdly, the diet gives far more nourishment than can be introduced into the body by nutrient enemas, and is, therefore, more desirable in patients who have frequently been for a long time in a state of semi-starvation, or have suffered a loss of blood, or both.

Fourthly, that in cases treated by this method rectal injections may be entirely avoided. This is an advantage in a hospital, and a still greater advantage in treating cases at their homes, where rectal injections are not only regarded as extremely unpleasant, but are seldom efficiently administered.

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Table I—Thirty-three Cases treated by the Lenhartz Diet.

Case No.	Name, age, occupation, hospital No.	Date of admission	Previous history	Duration of symptoms, this attack	Symptoms	HEMORRHAGE		Anemia, teeth, &c.	Treatment	Duration of pain in hospital	Time till on full diet	Time in hospital	Convalescent hospital	Weight	Subsequent history
						How often and much before admission	Reurrence under treatment								
1	L. P., 25, housemaid, 1516	Oct. 5, 1906	In-patient 3 years ago with gastric ulcer, anemic since	14 days	Pain, vomiting, tenderness in epigastrium	1 oz.	—	Anemic, decayed teeth	Saline enemata for 3 days, then Lenhartz diet	3 days	25 days	35 days	—	Gained 4½ lb. in last 10 days	Recurred; see below, Case 5
2	L. D., 24, housemaid, 1575	Oct. 18, 1906	Pneumonia, chorea, rheumatism, nephritis, gastric ulcer, with hæmorrhage 2 years ago	Worse for 1 month	Pain, vomiting, hæmorrhage	½ pt.	—	Albuminuria; Hb 60 per cent.	Lenhartz diet	Diminished gradually for 3 weeks	Fish in 23 days, fish diet in 40 days	43 days	—	Lost 6 lb.	Two attacks since
3	B. S., 17, shop assistant, 1581	Oct. 19, 1906	Same symptoms 4 months ago	14 days	Pain, vomiting, tenderness	No	—	Hb 87 per cent.	Lenhartz diet	3 to 4 days	19 days	22 days	—	Gained 3½ lb.	One attack since
4	M. B., 22, dress-maker, 1838	Dec. 7, 1906	In-patient in Mar., 1906, off for 8 months with gastric ulcer	On and off for 8 months	Pain, vomiting, tenderness	1½ oz.	—	Very nervous, moderate anemia	Lenhartz diet	1 month, occasionally	2 months	67 days	—	Lost 4 lb.	See Table II, Case 21

5	L. P., 25, house- maid, 1884	Dec. 18, 1906	See above, Oct. 5, 1906	5 weeks	Pain, vomit- ing, tender- ness	—	—	Hb 92 per cent., R. B. C. 5,100,000	Lenhartz diet; teeth extracted while in hospital	8 days; 5 weeks, 3 days	2 weeks	Gained 34 lb. in hos- pital	—	
6	E. F., 27, parlour- maid, 1905	Dec. 22, 1906	In-patient 2 years ago with gastric ulcer and hæmorrhage	14 days	Pain, vomit- ing, tender- ness	1 pt.	—	Hb 93 per cent., R. B. C. 4,772,000; alcoholic	Lenhartz diet	2 days	28 days	49 days	Yes	Return of sym- ptoms
7	A. H., 24, married, 73	Jan. 10, 1907	Pleurisy, pneumonia, gastric pain attack 6 months ago	16 days	Pain, vomit- ing, tender- ness	2 or 3 times, 4 oz.	—	Hb 85 per cent.	Milk and saline en- mata 3 days, calcium chloride <i>per rectum</i> , nutrients; Len- hartz diet third day	Not recorded	Fish diet 18 days	21 days	—	No return of gastric ulcer; pneu- monia since
8	E. K., 32, married, 80	Jan. 10, 1907	—	3 days	Hæmorrhage	$\frac{1}{2}$ pt.	—	Hb 80 per cent.	Nutrient enemata 2 days, then Lenhartz diet	Not recorded	Fish diet in 14 days	26 days	—	—
9	M. B., 25, cook, 184	Feb. 1, 1907	Anæmia, dyspepsia	1 day	Hæmorrhage	1 pt.	—	Hb 65 per cent.	Lenhartz diet	3 days	17 days	38 days	Yes; Gained Hb 80 per cent.	—
10	E. K., 18, servant, 217	Feb. 7, 1907	Scarlet fever 6 months ago	6 mon.; dyspep- sia 2 yrs.	Pain, vomit- ing, tender- ness	4 $\frac{1}{2}$ pt.	—	Hb 65 per cent., nervous	—	None recorded	19 days	20 days	Yes	Lost 1 lb. April, 1907, rheumatic fever with endocar- ditis; symptoms of ulcer in October; no hæmorrhage; recovery with milk diet

Case	Name, age, occupation, hospital No.	Date of admission	Previous history	Duration of symptoms this attack	Symptoms	Hæmorrhage		Anemia, teeth, &c.	Treatment	Duration of pain in hospital	Time till on full diet	Time in hospital	Convalescent hospital	Weight	Subsequent history
						How often and	Recurrence under treatment								
11	C. W., 50, misioner, 262	Feb. 15, 1907	Influenza, gastric ulcer 2 or 3 years ago	4 weeks	Pain, vomiting, tenderness	Once, little	—	Very neurotic	Lenhartz diet	None for 9 days, then some which passed off	25 days	26 days	—	Lost 1 lb.	Well since
12	A. E., 32, married, 301	Feb. 25, 1907	6 years pelvic pain following confinement	1 week	Hæmorrhage	1 pt.	—	Artificial teeth; Hb 74 per cent., R.B.C. 3,980,000	Lenhartz diet	No gastric pain	34 days	Hb 81 percent., R.B.C. 4,244,000 41 days	—	Lost 4½ lbs.	—
13	A. H., 28, kitchen-maid, 481	Mar. 28, 1907	Some similar but milder attacks	14 days	Pain, vomiting, tenderness	2 or 3 times, little	—	Anæmic	Water injections 2 days, then Lenhartz diet	Not recorded	Fish diet in 25 days	35 days	—	—	—
14	L. B., 31, dress-maker, 716	May 15, 1907	Been in twice for gastric ulcer in last 15 months	Severe for a week	Pain, vomiting, tenderness	—	—	Not obviously anæmic	Lenhartz diet begun on third day, then vomiting improved, but pain recurred on sixth day; posterior gastrojejunostomy performed 36th day; ulcer on posterior wall; recovered	Pain and vomiting	65 days	69 days	—	lost 5 lb.	—

15	A. C., 22, servant, 756	May 22, 1907	Scarlet fever followed by diphtheria 12 years ago; gastric ulcer with hemor- rhage 12 months ago	14 days	Pain, hemor- rhage, ten- derness	Once, $\frac{1}{2}$ oz.	—	Anæmic; Hb 75 per cent.	Lenhartz diet	2 days	25 days	47 days; Hb 95 per cent. on six- teenth day	—	gained 7½ lb.	Indiges- tion last 3 months, not left work
16	E. O., 20, 813	June 3, 1907	Anterior gas- trojejun- ostomy for bounddown perfora- tion of gas- tric ulcer 5 months ago; pain returned 14 days after leav- ing conval- escent hospital	2 months	Pain, vomit- ing, tender- ness	Hæ- mor- rhage in former attack	—	Hb 85 per cent.	Lenhartz diet	6 days	24 days	34 days	Yes	gained 6½ lb.	Well
17	C. P., 19, theatre attend- ant, 831	June 6, 1907	Appendicitis; operation in 1906	3 weeks	Pain, vomit- ing, tender- ness	2 oz.	—	Anæmic	Lenhartz diet	None recorded	Fish diet in 19 days	28 days	Yes; 1 month	—	—
18	M. H., 26, 910	June 23, 1907	Similar at- tack 9 months ago with hæ- morrhage	2 days	Very severe vomiting, pain, ten- derness	—	—	Anæmic, many carious teeth and stumps	Peptonized milk 4 days, then Lenhartz diet	Not recorded	Pounded fish in 16 days	24 days	—	—	—
19	G. B., 23, ward- maid, 984 (Case 16 in Table II)	June 20, 1907	Been treated three times by other methods, anterior gastrojejun- ostomy	14 days	Pain in stomach, vomiting, also drag- ging pain about rear	—	—	—	Peptonized milk 1 day, then water enemata 5 days, then Len- hartz diet	Not recorded	Pounded fish in 16 days	27 days	Readmission, 1907; severe vomiting or hæmor- rhage; treated gradually milk diet; left hospital unrelieved, recovered at Conval- escent Hospital	October, 1907; severe pain, no vomiting or hæmor- rhage; treated gradually milk diet; left hospital unrelieved, recovered at Conval- escent Hospital	

Case	Name, age, sex, occupation, hospital No.	Date of admission	Previous history	Duration of symptoms	Symptoms	HEMORRHAGE		Anemia, teeth, &c.	Treatment	Duration of pain in hospital	Time till on full diet	Time in hospital	Convalescent hospital	Weight	Subsequent history
						How often and much before admission	Recurrence under treatment								
20	A. R., 23, kitchen-maid, 1242	Sept. 10, 1907	Indigestion some months	2 days	Pain, vomiting, tenderness	—	—	Anemic, carious teeth	Milk, eggs, bread and butter 3 days, then Lenhartz diet	10 days, less after Lenhartz diet begun	Fish diet in 20 days	29 days	Yes	—	Well
21	A. L., 25, parlour-maid, 1374	Oct. 7, 1907	Gastritis 7 years ago	14 days	Pain, vomiting, tenderness	4 oz.	—	Anemic; fourteenth day Hb 55 per cent.; eighteenth day Hb 70 per cent.	Lenhartz diet	2 days	Fish diet in 10 days	23 days	Yes	—	—
22	R. A., 34, house-keeper, 1562	Nov. 17, 1907	Glands in neck, neuralgia	14 days	Pain, hemorrhage, tenderness	1 pt. on third day	1 pt. on third day	Anemic; Hb 75 per cent.	Lenhartz diet without ice-bag till hemorrhage, was not lying flat when hemorrhage came on, then saline and nutrient enemata 10 days, then graduated milk diet; melena for 4 days after this	—	Fish diet in 30 days	36 days	—	—	—
23	M. T., 16, dress-maker, 525	May 5, 1908	—	5 days	Pain, hemorrhage	Twice, 4 oz. on day of admission	—	Blanched, pulse 124	Saline enemata 1 day, then Lenhartz diet	7 days	Fish diet in 18 days	36 days	Yes; 14 days	—	Well

24	M. S., 31, 676	June 4, 1908	Gastric ulcer 6 months 4 years ago with ha- morrhage	Pain, tender- ness	Three times, largest 1 pt.	—	—	Milk one day, then Lenhartz diet	Less than 5 days	Fish in 15 days	36 days	12 days	—	Well
25	J. B., 34, gun- smith, 1020	Aug. 14, 1908	Severe gastric ulcers with hemor- rhage in Aug., 1906 and 1907, and April, 1908	14 days Pain, hemor- rhage	1 pt. 1/2 pt.	—	Hb 60 per cent.	Lenhartz diet	2 to 3 days, re- curred once on tenth day	20 days	32 days	14 days	Gained 24 lb. in hos- pital	Well; has gained 11 lb.
26	B. W., 38, married, 1087	Aug. 28, 1908	Six children	14 days Pain, vomit- ing, tender- ness	1 pt. on 27th	—	Pale, carious teeth	Saline enemata 3 days, then Lenhartz diet, horse serum	More than 4 days	Fish diet in 21 days	26 days	21 days	—	Well, oc- casional indiges- tion at- tributed to teeth
27	J. N., 20, house- maid, 1122	Sept. 5, 1908	Five months, dyspepsia	14 days Pain, vomit- ing severe, tenderness	A little on second day	—	Pale, arti- ficial teeth	Lenhartz diet	19 days	Pounded fish in 19 days	32 days	28 days	—	Pain and vomiting last 14 days
28	H. S., 33, 1253, male	Oct. 2, 1908	Nine months, dyspepsia	— Pain, hemor- rhage	1 1/2 pt. on second day	—	Anæmic	Modified Lenhartz for 23 days with success, then seen by sur- geon who did pos- terior gastrojejun- ostomy, 11 days later vomiting and dilated stomach, gradual improvement	7 days	Pounded fish in 13 days	54 days	21 days	—	Well
29	E. O., 18, house- maid, 1292	Oct. 9, 1908	Several months, dyspepsia	3 months Pain, vomit- ing, hemor- rhage	Sev- eral times, the last 14 days before admis- sion	—	—	Lenhartz diet	10 days	17 days	19 days	—	—	In hospi- tal for recur- rence, very mild attack

Case	Name, age, occupation, hospital No.	Date of admission	Previous history	Duration of symptoms this attack	Symptoms	HEMORRAGE		Anemia, teeth, &c.	Treatment	Duration of pain in hospital	Time till on full diet	Time in hospital	Convalescent hospital	Weight	Subsequent history
						How often and much before admission	Recurrence under treatment								
30	A. G., 15, housemaid, 1106	Sept. 2, 1908	Similar attack 6 months ago. Ill 5 weeks	1 day	Pain, vomiting, hemorrhage	Once much before admission	—	Carious stumps	Lenhartz diet	3 days	Fish diet in 12 days	15 days	—	—	Well
31	L. B., 24, servant, 1668	Dec. 30, 1908	—	3 months	Pain, vomiting, tenderness	—	—	—	Saline enemata for 3 days, then 6 days graduated milk diet, still pain and discomfort. Lenhartz diet begun and continued with success	—	Patient vegetarian and no fish or meat was taken	21 days	—	—	—
32	M. T., 46, cook, 12	Jan. 3, 1909	Dyspepsia 3 years ago and at intervals since	5 weeks	Pain, vomiting, hemorrhage	Twice, 1 pt. the first time since	—	Hb 66 per cent.	Lenhartz diet	3 days	—	—	—	—	—
33	K. L., 36, housekeeper, 66	Jan. 13, 1909	Occasional severe pain for 4 years	18 days	Pain, vomiting, hemorrhage	Three times profuse, 3 qt. mentioned, last hemorrhage 13 days ago	—	Blanching, conjunctiva and gums	Been carefully treated by doctor 13 days before admission by nutrient enemata and graduated diet; Lenhartz diet	No pain	—	—	—	—	—

Table II—Thirty-four Consecutive Cases treated by other Methods.

Case	Name, age, occupation, hospital No.	Date of admission	Previous history	Duration of symptoms this attack	Symptoms	HEMORRHOGE		Anemia, teeth, &c.	Treatment	Duration of pain	Time till on full diet	Time in hospital	Convalescent hospital	Weight	Subsequent history
						How often and how much this attack	Recurrence under treatment								
1	A. B., 30, servant, 1550	Oct. 11, 1906	Gastric ulcer 10 years ago with hæmatemesis	7 months	Pain, vomiting, tenderness	—	—	Artificial teeth	Albumin water 6 days, milk 10 days; refused operation; after 2 months began to improve	—	Fish diet in 59 days, vomiting still a little	92 days	—	—	Posterior gastro-enterostomy done later
2	S. B., 24, dress-maker, 1591	Oct. 22, 1906	Similar attacks for 9 years	8 weeks	Pain, localized, vomiting	Black vomit	—	Anæmic	Nutrient and saline enemata 7 days, then graduated milk diet	6 days	Pounded fish in 14 days	16 days	Yes	—	—
3	A. N., 35, lady's maid, 1699	Nov. 12, 1906	Similarly attacked 8 years ago, hæmorrhage 5 years ago	14 days	Pain, vomiting, tenderness	—	—	Artificial teeth	Fluid foods from the beginning	—	Pounded fish in 12 days	23 days	Four attacks of pain and vomiting; does work now	—	Four attacks of pain and vomiting; does light work now
4	A. D., 34, servant, 1712	Nov. 14, 1906	Pneumonia, bronchitis, sore throats, piles	21 days	Pain, vomiting, tenderness	Twice, 2 dr.	—	—	Saline injections 1 day, then milk only a week	16 days	Fish diet in 10 days	21 days	Yes	—	—
5	E. L., 27, cook, 1739	Nov. 21, 1906	Gastric ulcer with hæmatemesis 5 months ago	9 weeks	Pain, vomiting	Twice, 3-4 oz.	—	Anæmic	Nutrient enemata 4 days, then graduated milk diet	3 days, returned 7 days later, when fish was tried	30 days	49 days	Yes	—	—

Case	Name, age, occupation, hospital No.	Date of admission	Previous history	Duration of symptoms	Symptoms	HEMORRAGE		Anemia, teeth, &c.	Treatment	Duration of pain	Time till on full diet	Time in hospital	Convalescence hospital	Weight	Subsequent history
						How often and how much this	Recurrence under treatment								
6	A. P., 25, dress-maker, 1906	Dec. 22, 1906	Gastric ulcer 6 months ago	6 weeks	Pain, vomiting, tenderness	—	—	Anemic, displaced uterus	Milk, soup, albumin water from beginning; 3 days later nutrient enemata	6 days	23 days	25 days	Yes	—	Well
7	M. K., 33, waitress, 104	Jan. 16, 1907	Influenza, less severe attacks of pain during past 2 years	1 week	Pain, vomiting, tenderness	1 dr.	—	Anemic, moderate	Peptonized milk by mouth, water by rectum	9 days	Fish diet in 15 days	21 days	—	—	—
8	P. W., 21, house-maid, 114	Jan. 18, 1907	Dyspepsia, 1 year	2 months	Pain, vomiting, tenderness	—	—	Hb 80 per cent.	Peptonized milk 1 day, then water and nutrient enemata 5 days, then graduated milk diet	—	Fish diet in 24 days	32 days	Severe indigestion at times; subacute symptoms most of the time since leaving hospital	—	—
9	S. R., 26, cook, 135	Jan. 23, 1907	Been in Brompton Hospital with chest	3 weeks	Vomiting, tenderness	4 times	—	Hb 65 per cent.	Water and nutrient enemata 3 days, then graduated milk diet	—	Fish diet in 15 days	22 days	Now in Hospital with another attack	—	Returned to hospital 3 months later; 1908, recurrence with hemorrhage, posterior gastro-enterostomy, July 3, followed by bronchitis; still has frequent vomiting and has done no work yet
10	K. S., 29, cook, 202	Feb. 4, 1907	Gastric ulcer 7 years ago	3 weeks indigestion 6 months	Pain, coffee ground vomit, tenderness	Twice	—	—	Nutrient enemata 4 days, then graduated milk diet	4 days	Fish diet in 16 days	31 days	Returned to hospital 3 months later; 1908, recurrence with hemorrhage, posterior gastro-enterostomy, July 3, followed by bronchitis; still has frequent vomiting and has done no work yet	—	—

11	M. B., 37, married, 250	Feb. 13, 1907	In hospital with gas- tric ulcer 2½ years ago	5 weeks	Pain, vomit- ing tender- ness	Once, a little	—	—	Peptonized milk 2 days, then water enemata 3 days; then graduated milk diet	Not recorded	Pounded fish in 16 days	21 days	—
12	A. M., 23, servant 274	Feb. 20, 1907	Vesical cal- culus, gas- tric ulcer 2 years ago	6 months	Vomiting, pain	Once, 1 oz.	—	—	Graduated milk diet begun at once	Not recorded	Fish diet in 16 days	21 days	Return of sym- ptoms
13	F. A., 28, dress- maker, 389	Mar. 11, 1907	Anemia at 17	Pain 6 months	Hemorrhage	Three times large clots	—	—	Nutrient enemata 5 days, then graduated milk diet	Not recorded	Fish diet in 17 days	23 days	Well
14	A. J., 35, 431	Mar. 20, 1907	Similar at- tacks on and off 5 years	1 month	Pain, vomit- ing, tender- ness	—	—	—	Water and nutrient enemata 6 days, then milk 3 days, then enemata again and graduated diet	14 days	Fish diet in 26 days	41 days	—
15	H. R., 37, servant 537	April 8, 1907	Gastric ulcer with hem- orrhage, 6 and 2½ years ago	5 weeks	Pain, vomit- ing, tender- ness	Once, little	—	—	Abscess in mouth while in hospital	41 days	Fish diet in 49 days	91 days	—
16	G. B., 23, 640 (Case 19 in Table I)	April 30, 1907	Gastric ulcer 1 year ago in this hos- pital; re- curred and anterior gastrojejun- ostomy done at another hospital	2 weeks	Pain, vomit- ing, tender- ness	5 oz.	—	—	Water enemata 7 days, 12 days, then graduated diet; recurred on 27th day put on 26th day back on enemata	12 days, recurred on 26th day	Fish diet (second time) in 36 days	43 days	—

Case	Name, age, occupation, hospital No.	Date of admission	Previous history	Duration of symptoms this attack	Symptoms	HEMORRHAGE		Anemia, teeth, etc.	Treatment	Duration of pain	Time till on full diet	Time in hospital	Convalescent hospital	Weight	Subsequent history
						How often and how much this attack	Reurrence under treatment								
17	L. N., 22, servant, 667	May 5, 1907	Gastric ulcer 6 months ago	5 weeks	Pain, vomiting, tenderness	Once, little	—	Nervous, Hb 70 per cent.	Peptonized milk from beginning and graduated milk diet	12 days	Fish diet in 9 days	18 days	—	—	—
18	E. D., 35, married, 741	May 20, 1907	Severe dyspepsia 6 years ago, and occasionally since	4 days	Pain, vomiting, tenderness	Once, 2 pt.	—	Blanching	Nutrient enemata 4 days, then milk 2 days, then returned to nutrient enemata, 2 days, then graduated milk diet	11 days, again on twenty-third day	Fish diet in 18 days	31 days	Yes	—	Severe pain, no vomiting; has kept at work
19	K. G., 24, kitchen-maid, 780	May 28, 1907	Pneumonia, scarlet fever, anemia, since aged 14	1 day	Pain, occasional vomiting, hæmorrhage	Profuse	—	Carious teeth	Nutrient enemata 3 days, water by mouth 1 day, then graduated milk diet	Not recorded	Fish diet in 16 days	29 days	Yes	—	—
20	F. P., 20, 980	June 3, 1907	Dyspepsia	1 day	Hæmorrhage, pain, tenderness	5 oz.	16 oz. on 2nd day, 12 oz. on 3rd day	Very anemic; artificial teeth	Water enemata 10 days, then graduated milk diet, double parotitis fourth to eleventh days	Not recorded	Fish diet in 32 days	36 days	—	—	Well
21	M. B., 23, dress-maker, 1126 (vide Table I, Case 4)	Aug. 10, 1907	Gastric ulcer treated by Lenhart, 1906; in again Mar., 1907, fifth period, as in-patient	6 weeks	Pain, vomiting	A little	—	Very neurotic, ulcer of leg, carious teeth	Milk and soda 11 days, then graduated diet 13 days, then saline enemata 2 days, followed by graduated diet	25 days	Fish diet in 27 days	34 days	Return of symptoms; three operations since; now in West London Hospital with recurrence	—	—

22	E. M., 42, 1583	Nov. 21, 1907	Nephropexy 6 years ago	1 week	Pain, hæmor- rhage	Twice, more than 2 pt. each time	—	Carious teeth	Small quantities of milk from beginning with horse serum, saline enemata 7 days	2 days	Fish diet in 10 days	33 days (tonsil- litis during conva- lescence)	—	
23	B. S., 46, cook, 1634	Nov. 30, 1907	—	3 weeks	Pain, hæmor- rhage	1 pt.	Me- læna, 9 days	—	Saline and nutrient enemata 10 days, then graduated milk diet	2 days	Fish diet in 18 days	32 days	Yes; 14 days	—
24	M. S., 28, house- maid, 1694	Dec. 13, 1907	Indigestion	2 months	Pain, severe, hæm or rhage, and melæna	1 pt.	—	Anæmic	Saline and nutrient enemata 11 days, then graduated milk diet	Not recorded	Fish diet in 20 days	40 days	Yes; 26 days	Well; oc- casional indiges- tion
25	A. K., 26, house- maid, 1711	Dec. 18, 1907	Attack of epi- gastric pain 3 years ago for 6 weeks	1 week	Pain, hæmor- rhage	Over 1 pt. 5 days ago	—	Hb 35 per cent.	Graduated milk diet from beginning	Not recorded	Fish diet in 9 days	18 days	—	Well
26	L. M., 30, cook, 1736	Dec. 23, 1907	—	1 day	Pain, hæmor- rhage	Over 2 pt.	—	Blanched running pulse	Nutrient and saline enemata; albumin water by mouth 3 days, then pepton- ized milk by mouth	—	Died on	fourth day	—	—
27	J. F., 22, servant, 1749	Dec. 25, 1907	Indigestion 2 years	3 weeks	Pain, vomit- ing, hæ- morrhage	Over 1 pt.	Over 1 pt. 3rd day	—	Water enemata, third day peptonized milk, this vomited, and nutrient enemata 10 days; then gradu- ated milk diet	8 days	Fish diet in 33 days	35 days, delayed by tonsil- litis; (scarlet fever)	Yes	Well

Case	Name, age, occupation, hospital No.	Date of admission	Previous history	Duration of symptoms this attack	Symptoms	HEMORRHAGE		Anemia, teeth, &c.	Treatment	Duration of pain	Time till on full diet	Time in hospital	Convalescent hospital	Weight	Subsequent history
28	E. M., 24, kitchen maid, 40	Jan. 8, 1908	Similar attack 7 months ago; treated 3 months in hospital and convalescent hospital	14 days	Pain, vomiting, tenderness	How often and how much this attack	Recurrence under treatment	Pale, artificial teeth, Hb (7th day) 81 per cent.; constipation	Nutrient enemata 10 days, 10 c.c. horse serum by mouth, graduated milk diet	9 days and later	25 days	65 days; (temperature rose and pains recurred for a day or two later, and delayed discharge)	Recurred three months after leaving hospital since	one leaving other	month hospital; attacks
29	L. H., 24, house-parlour maid, 97	Jan. 18, 1908	—	1 month	Pain, hemorrhage, tenderness	Once	—	Blanching	Nutrient enemata 4 days, then graduated diet added	Not recorded	Fish in 21 days	32 days	Yes; 3 weeks	—	—
30	R. J., 24, cook, 165	Feb. 1, 1908	—	3 months	Pain, vomiting, tenderness	Once, ten-a little	—	Anæmic	Nutrient enemata 9 days, then milk	4 days	Fish in 20 days	92 days	—	—	—
31	L. B., 37, married, 188	Feb. 5, 1908	Gastric ulcer 2½ years ago	1 day	Pain, vomiting, hemorrhage	½ pt.	5 oz. third day	Well nourished; pregnancy 3 months	Water and nutrient enemata, and Ca. chl. 7 days, then graduated milk diet (parotitis on tenth day)	Less than 5 days	Fish in 14 days	25 days	—	—	Well

32	J. H., 20, kitchen- maid, 247	Mar. 4, 1908	Dyspepsia	3 months	Pain, tender- ness, he- morrhage	Once, 3 weeks ago	—	—	Milk 1 day, then saline enemata 4 days, then graduated diet	Less than 5 days	Fish in 12 days	29 days	—
33	F. O., 20, house- keeper, 269	Mar. 9, 1908	Gastric ulcer 2 years ago	1 day	Pain, hemor- rhage	1 pt.	A little on second day with vomit- ing	Anaemic, carious stumps	Nutrient enemata 2 days, then gradu- ated milk diet	Less than 9 days	10 days	24 days	—
34	A. T., 21, 314	Mar. 18, 1908	Gastric ulcer 2 years ago	5 months	Pain, vomit- ing, tender- ness	Sev- eral times small quan- tity	—	Anaemic	Milk 1 day, then nu- trient and saline enemata 5 days, then graduated diet	—	Fish diet in 25 days	31 days, on 28th day Hb 85 per cent.	—

DISCUSSION.

Dr. LANGDON BROWN said he thought Dr. Spriggs had done a service in emphasizing the importance of that very practical method of treatment. His experience with it, now based on fourteen cases, had been very favourable. Rectal feeding was admittedly unsatisfactory; apart from the difficulties, its efficacy could be seriously questioned. Dr. Sharkey, in his lecture before the College of Physicians in 1906, stated that in his series of cases of rectal alimentation he got as much as 75 per cent. of the nutrient matter absorbed, but the method adopted was simply to wash out the bowel afterwards and estimate the amount of nitrogen recovered, the rest being regarded as absorbed. That was not a safe method, because it was very difficult to recover all the nitrogen by simple washing out. He had been impressed by a case recorded by Dr. Herringham in proof of that. The patient was a medical man, and was careful to see that the washings were properly carried out. Ten days from the first rectal feed he passed an enormous and very disgusting evacuation, and the patient's comment was that if he had been retaining all that in his intestines it could not have been doing him much good. A better estimation of the absorption was afforded by the amount of nitrogen in the urine. The output of nitrogen by the urine when the patient was on rectal feeding was the same as that of a patient being starved. Laidlow and Ryffel showed that the amount of nitrogen in the urine on rectal feeding was the same as that in the professional faster at about the fifteenth to the twentieth day. Dr. Langdon Brown had estimated the urine in a patient who, being on salines only, was showing the same nitrogen output as any fasting person. Then for two days he gave the ordinary nutrient enemata, 4 oz. of milk with a drachm of plasmon and a drachm of sugar, given every four hours, after pancreatizing. During those two days the nitrogen continued to decrease. Then he gave the same quantity of milk and plasmon by the mouth, and at once the nitrogen output began to rise. This result threw grave doubt on the power of the rectum to absorb food. And as soon as the efficacy of rectal feeding began to be doubted, its obvious disadvantages assumed more significance. He had seen that persistent vomiting, to which the author had referred, several times in association with rectal feeding, which ceased on feeding by the mouth. He thought it was sometimes the vomiting of acidosis, because at such times diacetic acid might be found in the urine. Parotitis was sometimes a serious complication in cases on rectal feeding, but it could often be obviated by giving a bismuth lozenge to suck. He did that for some time before adopting the Lenhartz method, and parotitis had not occurred in any of these cases. By sucking the lozenge the flow of saliva was maintained, and the alkaline products taken into the stomach would tend to neutralize any gastric juice which might be secreted, either as a result of the thought of food, or as the result of rectal feeding. He objected to the use of glycerine as a mouth-wash, believing that the resulting

desiccation helped in the production of parotitis. It appeared from recent work on autolysis that digestion went on more rapidly in fasting tissues than in well-fed tissues, and as rectal feeding was partial starvation, it was not to be recommended in people who had been much depleted by repeated hæmorrhages, and whose stomachs were virtually undergoing self-digestion. In two cases he did not have a very good result from the Lenhartz treatment, but they were both Jews, and very neurotic. One had a recurrence of the hæmorrhage, but when he was put on the orthodox treatment he had another recurrence. Therefore the Lenhartz method could not be held answerable for the bleeding. The other patient, who was also neurotic, said that the diet gave her pain, but he did not regard that as altogether due to the stomach, as she had some colitis. One of the most satisfactory cases was in a man, aged 33, who had four hæmorrhages before coming into hospital, and melæna after admission, so that there was no doubt about the bleeding. He improved rapidly under the Lenhartz treatment, and was very comfortable, presenting a striking contrast to the patients who were always grumbling on the orthodox method. He slightly modified Lenhartz's directions. He did not think it advisable to give raw meat so soon as did Lenhartz; it being a stimulant of the gastric juice, he believed it tended to cause the return of pain. Neither did he give raw ham, which was not much relished by English people; he gave minced chicken instead. The advantages of the treatment, especially in private practice, were great, and he was glad Dr. Spriggs had called attention to it.

Dr. LAURISTON SHAW said that he was pleased that a more rational mode of treatment was being considered. He had never been a strong upholder of the orthodox method of prolonged starvation. He did not treat his gastric ulcer cases by the heroic measures which some physicians thought necessary. The chief reason he hailed the less drastic method was that gastric ulcer patients frequently tended to become highly neurotic. He believed a fair number of supposed cases of gastric ulcer were really neurotic dyspepsias. But whether the patient was already only neurotic, or whether she suffered from gastric ulcer, nothing was more likely to induce a feeling of great trepidation at her condition than to be surrounded by medical people who themselves seemed to be in a similar trepidation as to a recurrence of the hæmorrhage or other symptoms. In the out-patient department the neurotic condition of patients who had been previously treated in hospital once or twice by prolonged starvation was very marked. He hoped the Lenhartz method would receive wider adoption in this country.

Dr. AULD said the opener seemed to have carried out the Lenhartz method without regard to the class of case he was dealing with; in opposition to which he counselled treating every individual case as a separate entity, to be specially studied. Would Dr. Spriggs treat an elderly man who had had gastric hæmorrhage exactly the same as he would treat a young girl, or a full-blooded woman the same as an anæmic boy? To treat a series of cases according to a rigid method was not scientific. It was fully realized that in such cases it was necessary for the doctor to feel his way. One patient might be able safely to

take a little semi-solid food, which in another patient would bring on bleeding. He had rarely had recourse to rectal feeding in such cases; he had very good results on a sort of modified Lenhartz diet. He did not as a rule attach much value to statistics. Dr. Spriggs had put forward a list, but some of the cases might be simply due to neurosis of the gastric mucous membrane. (Dr. SPRIGGS: All except four had hæmorrhage.) It had not been proved that the hæmorrhage was from ulcer of the stomach, and gastric ulcer could not be diagnosed with certainty from hæmorrhage and pain. And he did not think it fair that the thirty-four cases should be contrasted with those which had nutrient enemata. It would be fairer to present a list of those treated by the mouth, but not necessarily by the Lenhartz method. He objected to rushing the diet, because that encouraged recurrence, even though it might not be at once.

Dr. B. G. MORISON said that although the Lenhartz method was new, the principle which underlay it was not new. He held an intermediate position in regard to the treatment of gastric ulcer; he did not quite believe in the Lenhartz treatment, neither did he repudiate treatment by enemata. The Lenhartz method was admissible in subacute and chronic cases, but to begin it soon after a recent hæmorrhage was a precipitate course. If the material for the nutrient enema was properly prepared and predigested, some good must ensue from it. After a hæmorrhage he thought two days should elapse before anything was given by the mouth; resorting to rectal feeding, which had the advantage of giving the stomach a rest. His habit was to give $\frac{1}{2}$ pint at a time, not very frequently, say every four hours. After the two days, small quantities could be given by the mouth. Solid food should not be given for weeks, and carbohydrates should be given in the most assimilable form. He had not met with the difficulties regarding enemata in private practice which had been mentioned.

Dr. BASIL PRICE asked whether Dr. Spriggs regarded the giving of bismuth, referred to by him, as an important accessory in the treatment of gastric ulcer. Taking the ordinary symptoms in proof of the existence of a gastric ulcer, he had had eight cases in which he gave no drug by the mouth, and there was no recurrence of hæmorrhage or other symptoms. He waited forty-eight hours in two or three cases before introducing food by the mouth, and he had had satisfactory results in all. On two occasions he had the advantage of a view of the stomach after the giving of bismuth. The bismuth had apparently gravitated to the fundus of the stomach, and the pyloric area, where the ulcer was situated, was in a somewhat contracted condition; the bismuth seemed to have exercised no protective action on the ulcer. He wished to ask Dr. Spriggs's opinion in regard to a case in which there was a recurrence of gastric symptoms, such as hæmorrhage and melæna, after twelve months or more interval—this might not, of course, be due to the same ulcer; would he look upon that as an indication for surgical treatment, rather than again treating the case by the Lenhartz method?

Dr. H. C. CAMERON said the Lenhartz method was not the only rival in the field, nor the only substitute for rectal feeding. Another method, which

had the approval of physiologists, was treatment by a diet mainly or entirely composed of fat. He had seen at Guy's Hospital two cases treated upon a diet of olive oil and cream, and both did extremely well. On a fat diet there was the smallest possible formation of hydrochloric acid. He thought Dr. Spriggs's statistics showed that some rational method of feeding was to be preferred to rectal feeding. To a certain extent he associated himself with Dr. Auld's remarks concerning statistics. The criticism which one must make in regard to a double series of statistics such as the present was that the morbid conditions in individual cases must differ so widely. At post mortems he had been struck by the different appearances of the ulcers; some were obviously acute and had been present only a few days; others had so much thickening and induration that they seemed to have existed for months, or even years. It seemed extraordinary that such varied conditions should be regulated by the same time-table. He believed there were two conditions—an acute ulcerative and a chronic ulcerative condition; and he asked Dr. Spriggs that he should indicate in his tables what was his conception of the morbid appearances in the stomach in each case. Recently he had looked up the histories of cases of acute ulcers which had perforated, and had found that it was exceptional to get a history of more than a day or two of dyspepsia. Such acute ulcers healed quickly under any rational form of treatment. In chronic cases with thickening and induration many months must elapse before a similar result was obtained.

Dr. J. GRAY DUNCANSON said there was one point of detail in the treatment to which he took exception—namely, giving chopped meat in cases of hæmorrhage, either from the stomach or the bowel. There was some analogy between the treatment of hæmorrhage of the stomach and that of the hæmorrhage which occurred in enteric fever. In the latter case he would not hesitate to give pounded or shredded raw beef at a very early stage, but he would strongly object to chopped or minced raw beef, or, what was worse, partially cooked, being given in ulcer of the stomach. Secondly, he considered, especially where there was anæmia, that fresh air and sunshine were of the utmost importance. He did not know whether that entered into the Lenhartz treatment.

Dr. SPRIGGS, in reply, said the giving of chopped meat in the cases he had observed did not cause recurrence of the hæmorrhage. He agreed as to the importance of fresh air; cases of gastric ulcer nearly always did well at the convalescent hospital. Some of the results of Senator's method were quite good. He (Dr. Spriggs) had not tried the olive oil treatment, but had read of a number of successful cases. In answer to Dr. Cameron, that gentleman would find that many of the cases on the list were not by any means up to the time-table. As far as he was able to judge, the two series comprised similar cases; and he had cut out cases in which heart, lung, or other trouble complicated the case. If there were any inequalities, the disadvantage was on the side of the Lenhartz treatment, because in them he had included some difficult cases. In answer to Dr. Auld, the patients were not treated by rule-of-thumb; in fact, very few were treated exactly alike—the treatment was modified

considerably to suit the cases. He would not invariably use Lenhartz's or any other method. He re-read a paragraph urging the importance of the physician using his common sense when at the bedside. Bismuth was certainly found sometimes to lie on the ulcer, but he thought its action was to cover up the general gastric mucous membrane, and, by diminishing the sensitiveness of the stomach as a whole, to render it less liable to contract suddenly. With regard to the recurrence of the ulcer, whether gastric ulcer was treated this way or that, or whether operation was done, many cases did recur, though whether from the same ulcer or not he did not know. On the other hand, complete healing often took place and many patients only had one attack. Lenhartz mentioned two cases which died from other causes during treatment, one on the fifteenth day, and the other on the eighteenth day, and the ulcers were found covered with mucous membrane. If a case of recurrence after one year were to come to him he would treat it again on the same plan. The liability to gastric ulcer seemed in many people, though by no means in all, to be limited to about five years. One speaker said it was precipitate to give Lenhartz treatment after a hæmorrhage. It might seem so, but about 300 cases now published showed that the treatment was especially successful in such cases. One could begin to put food into the stomach in the first twelve hours after a hæmorrhage, and in many cases it was given six hours after the bleeding. Dr. Langdon Brown's observations of the amount of nitrogen in the urine in rectal feeding were of great interest, and agreed with what most good observers found, that it was difficult to get very much food into the system via the rectum. Even if 75 per cent. of the nourishment were absorbed in the experiment quoted it was necessary to know how long that could be kept up. The cases he had narrated were not neurotic dyspepsias; for one thing, nearly all of them had hæmatemesis.

Therapeutical and Pharmacological Section.

March 2, 1909.

Dr. T. E. BURTON BROWN, C.I.E., President of the Section, in the Chair.

The Treatment of Spasmodic Asthma.

By CECIL WALL, M.D.

THE subject proposed for discussion by this Section this afternoon is one that has ever proved of interest to physicians and to physiologists, to empiricists and to theorists, to the orthodox and to the charlatan. Our knowledge of spasmodic asthma is far from complete, and the treatment of the disease is often one of the most difficult problems that the physician has to face. The uncertainty of our knowledge of the true nature of the disease has resulted in the recommendation of a vast number of remedies, so numerous, in fact, that a single practitioner can scarcely expect to have an opportunity of testing even a tithe of them. At a meeting such as the present the collective experience attempts to atone for the individual inexperience; my remarks must necessarily be eclectic. As Willis wrote with regard to spasmodic asthma in 1776, so may I now make preface. "It will help us in no wise to enumerate the varied drugs and prescriptions of which an immense forest may be found spreading through medical literature; it will suffice our purpose to discuss the principles of treatment and to detail some of the more favourite remedies for uncomplicated spasmodic asthma."

The principles of rational treatment must be based on theory, and unless the theory be correct the principles of treatment may be unsound, even though the actual practice may be satisfactory. In few diseases has theory played so large a part in suggesting treatment as in spasmodic asthma; unfortunately there is no unanimity of opinion even now with regard to the mechanism of an asthmatic attack.

It is almost universally admitted that during an attack of spasmodic asthma the lumen of the finer bronchial tubes is narrowed; the majority admit that in some way this narrowing of the bronchioles is effected through the intermediation of the nervous system; beyond this unanimity of opinion disappears. The theory which seems to find most support is a development of that originally suggested by Willis, adopted by Cullen, and put to the experimental test by C. J. B. Williams and recently by Brodie and Dixon; this theory supposes that the narrowing of the bronchial tubes is effected by contraction of the bronchial muscles under influences brought to them by the vagi nerves. The opposing theory supposes that the narrowing of the lumen of the bronchioles results from a turgescence of the lining membrane, such turgescence being due to vasomotor nerve influences, to local mechanical or chemical irritation, or to a local inflammatory process.

It is not my present purpose to discuss these rival theories; it suffices to point out that the distress of the asthmatic paroxysm is admitted to be due to the obstructed ingress and egress of the air to and from the pulmonary alveoli, and that any form of treatment which will reduce the obstruction in the bronchioles will give relief.

We may perhaps speculate still further upon the principles of treatment. Allowing that the supposition is correct that the narrowing of the tubes is brought about by the aid of nervous impulses, we may reasonably expect to give relief if we can prevent the origin of such impulses in the central nervous system, or their conduction along the nerves to the lungs. In the present state of our knowledge of spasmodic asthma, the object of prophylactic treatment must be the prevention of that form of activity of the central nervous system which results in efferent impulses leading to bronchiolar obstruction; the object of symptomatic treatment during the attack must be to widen the lumen of the bronchioles either by removing the turgidity of the mucous membrane if present, or by causing, directly or indirectly, a relaxation of the tone of the bronchial muscles.

Passing on from this brief statement of the general principles of treatment, I will detail some of the more favourite remedies for spasmodic asthma, and allude to the reasons, theoretical or empirical, which have led to their adoption.

First in order of importance are the measures prophylactic against the recurrence of attacks, and consequently, if successful, curing the disease.

All physicians admit the peculiar influence of climate and locality

in the production or prevention of paroxysms of asthma, and most will admit with Salter that the habit of asthma may sometimes be broken by residence in a suitable place. It is generally recognized that it is impossible to foretell the effect of a given climate or locality upon a given patient; on the whole, London has perhaps the best reputation as a health resort for the asthmatic, and, as Salter long since pointed out, the central parts seem better than the outskirts. The reason for this is not very obvious; explanations have been sought in the composition of the atmosphere, and it has been pointed out that since the days of Riverius and Culpepper sulphureous and fuliginous asthma specifics have been on the market. There may be another factor of even greater importance; I allude to the comparative constancy of the temperature in London as compared with the country. It is a familiar experience that a patient who seldom passes a night in his own house without an attack of asthma, even though it be in London, ceases to be troubled after admission to hospital. The chief change in the environment seems to be that the temperature of a hospital ward is not allowed to vary. If the asthmatic patient be also liable to attacks of hay fever, the importance of the locality of his residence very soon becomes obvious to him. I need not further enlarge upon the climatic treatment of asthma.

Just as all physicians agree that climate affects the asthmatic, so all agree as to the importance of attention to general hygiene and to the correction of any abnormality in the due performance of the natural functions of the body.

I will now turn to another aspect of the prophylactic treatment of asthma; avoidance of the exciting cause, if such can be found, by climatic or hygienic treatment cannot be considered as an entirely satisfactory cure of the disease. In 1871 Voltolini pointed out that there might be a connexion between nasal disease and asthma; since then much evidence has accumulated showing the importance of this connexion, and demonstrating that in some cases treatment of the nose results in the cure of asthma. Ten years ago, at a meeting of the Laryngological Society, Dr. Percy Kidd summed up the general opinion in the words: "if there was obvious disease of the nose, local treatment was advisable, though the uncertainty of the result as regards the asthma should be clearly explained to the patient." I think that this opinion still represents the views of the majority of physicians and rhinologists; it is in accordance with my own somewhat limited experience. I have notes, taken before and after operation, of twenty-three

cases in which the asthma was associated with some obvious disease of the nose. In fourteen of these some benefit was noticed, and in some the relief was considerable, though in none was a permanent freedom from asthmatic attacks recorded; in the remaining nine the tendency to asthma remained unchanged. I am fully aware of the fallacies inherent in statistics concerning hospital out-patients, but I quote them because they seem to bear out the received opinion of others.

In many patients who suffer from spasmodic asthma examination of the nose reveals no gross abnormality; the general consensus of opinion is that in such patients intranasal treatment is not likely to result in relief. In marked contrast to this view is that of Dr. Alexander Francis, who holds that the prognosis as regards the permanent relief of asthma by intranasal treatment is almost in inverse ratio to the amount of trouble found existing in the nose. Dr. Francis believes that "intranasal irritation is rarely the immediate cause of asthma, and that in the asthmatic state there exists a morbid connexion between the nose and the respiratory centre, which maintains the centre in such an unstable condition as to allow peripheral irritation to provoke an asthmatic reflex." He states that "he seeks by his method of treatment to restore the stability of the respiratory centre, and at the same time, as far as possible, remove all sensory irritation capable of exciting an asthmatic paroxysm." The treatment recommended is to draw a line with a galvanocautery point from a spot opposite to the middle turbinate body, forwards and slightly downwards, for a distance of rather less than half an inch. In a week's time the operation is repeated on the other side, and afterwards on alternate sides at intervals of ten days or a fortnight, as occasion requires. At the same time he recommends the ordinary method of treatment for any lesion or abnormality found existing in the nose, except in the case of mucous polypi, when cauterization of the septum should precede the removal of the polypi.

Dr. Francis gives records of over 400 cases treated by his method, and claims for it almost specific virtues. My own experience of the treatment is too small to enable me to express any opinion, but I hope that someone present in the room may be better able to pass judgment. In the few cases that I have seen treated according to Dr. Francis's directions, the desired result has not been obtained.

The remainder of my remarks upon the favourite remedies for asthma concern those methods of treatment which aim at the relief of symptoms rather than the cure of the disease. Certain drugs are reputed to render less frequent the attacks of asthma; those of which I

have the most practical experience are potassium iodide, stramonium, and arsenic. I believe that if the patient is troubled with frequent attacks of asthma he may be benefited sometimes by a course of potassium iodide in 3- or 4-grain doses, combined with an alkali. Arsenic may be added as a corrective, and occasionally seems to be of value in itself. In severe cases which persist in spite of these forms of treatment, I have found that stramonium, with or without potassium iodide, is often most useful. The best preparation is the extract, and it should be given in doses sufficient to produce slight toxic symptoms. Patients have told me frequently that in spite of the dry mouth and the difficulty in vision they prefer to continue taking the drug, because it helps them so much.

Other alkaloids of the atropine series have a similar effect: belladonna, hyoscyamus, and conium are sometimes substituted for or combined with stramonium. My own experience agrees with that of Salter, and leads me to suppose that the extract of stramonium made from the seeds is far superior to the tincture which is made from the leaves; the pharmacopœial dose of the extract is excessive. Most patients show toxic effects if they take $\frac{3}{4}$ gr. in the twenty-four hours. In one asthmatic patient, who was also the subject of gout, I found very marked relief following the use of colchicum; this is somewhat contrary to expectation, seeing that colchicine is said to have an action on the bronchial tubes allied to pilocarpine.

Of the utility of other drugs said to have a specific action in diminishing the tendency to asthma, I have but little experience. Of the drugs I have mentioned I deprecate the continued use, though I think that intermittent courses may be of value. Once only have I tried the effect of opium as a prophylactic: a girl who suffered as severely as any patient I have ever seen, and had experienced all forms of treatment without the slightest relief, improved in a most astonishing manner when small doses of compound tincture of camphor were ordered. Possibly this was merely idiosyncrasy.

The remedies for use at the time of an asthmatic attack are almost innumerable; time will not permit me to mention more than a few. Riverius and Culpepper recommend emetics for the treatment of the attack, and suggest tobacco water as one of the best; the virtues of tobacco were well known to these authors. "Tobacco taken in a pipe hinders the fit, so doth the leaf chewed, and also the smoke of cloves in a pipe." Fumes apparently were frequently employed by these physicians; many formulæ are given, and it is curious to note how

frequently sulphur is one of the ingredients. Willis repeats the recommendation of Riverius with approval. Cullen pinned his faith on opium. Emetics are not often used now, except in the case of children, when they may prove of great value. Salter pointed out that tobacco gave ease from its nauseating power, and was of little value in those habituated to its use; doubtless this is usually true, but some patients have told me that in spite of the habit tobacco persists in giving a measure of relief.

With regard to opium and its alkaloid morphia, it seems certain that we have in them the most potent remedy for the asthmatic paroxysm; the fear of creating a drug habit forbids their use except in the last extremity.

In 1802 General Gent introduced stramonium as a remedy for asthma, and from this time onward preparations of the solanaceous plants have formed the basis of many asthma specifics.

Whatever may be the explanation, there can be no doubt that the fumes of the stramonium powders give great relief to the majority of asthmatics. The simple formulæ containing stramonium, aniseed, and nitre are quite as efficacious, though perhaps less pleasant than the more complex, which may contain in addition lobelia, tobacco, tea, sulphur, eucalyptus or other ingredients. Nitre papers burnt in the room will sometimes help in the case of children, but in adults I have seldom known them give much relief.

Next in historical order of introduction is the acetone treatment; this was first used as an inhalant, in combination with iodine and hyposulphite of potash, by Dr. Churchill in the seventies; he kept secret the formula of his preparation, and its use fell into desuetude. Recently the formula has been published and trial has shown that in some cases, when other means have failed, this spray gives relief. The solution prepared according to Churchill's directions is inhaled as a vapour through the open mouth; as with other so-called cures, patients become habituated to its use, but it has the advantage of not being likely to engender a drug habit.

I am afraid that the same advantage cannot be claimed for some other proprietary specifics which have recently achieved popularity. These specifics, containing cocaine, atropine and a nitrite, are applied in the form of a spray or paint to the nasal mucous membrane. There is no doubt that a large number of patients find great relief from their use, but I believe that most will agree that cocaine even in dilute solutions is a dangerous drug to place in the hands of an asthmatic patient. The

use of these preparations bears out the theory which suggests the close relationship between the asthmatic tendency and the condition of the nasal mucous membrane. One of the later specifics of this class has substituted a solution of adrenalin for the cocaine, and therefore seems less objectionable.

Another group of remedies consists of those which affect directly the distribution of blood in the body. I have used amyl nitrite and nitroglycerine in some cases, and found sometimes considerable relief from the distress. One patient told me that a tablet of nitroglycerine taken after inhaling an asthma powder seemed to increase its effect markedly. In many patients, however, the headache produced by the nitrites is a disadvantage, which seems to outweigh the advantage.

Dr. Francis Hare, who is an advocate of the vasomotor theory of asthma, supports their use; he also points out that relief may follow if while warmth is applied to the body cold air is inhaled. Carrying the argument further, he has tried with success the local use of vasoconstrictors such as adrenalin or alum in a vapour combined with vasodilation by local applications externally.

In conclusion, I must apologize for the omissions which have been inevitable in a paper of this nature, and also for the necessary deduction that, in spite of all the investigations that have been made, it seems that in the treatment of asthma we must still regard each patient as a law unto himself.

DISCUSSION.

Dr. DIXON said he had not much that was new to lay before the Section on the matter, but would say a few words as to the way in which drugs acted in bronchial spasm. He held that there were two essential factors in asthma; there must be a certain type of medulla as a predisposing cause of the condition—a type known as neurotic. He showed some slides indicating the volume of the right lung of a dog, the amount of air passing in and out being measured. No anæsthetic must be given in those experiments, as it got down to the nerve-endings in the lung, and appeared to paralyse them, or at all events depressed them in such a way that the effect was unsatisfactory. The slide showed that on exciting the vagus a smaller amount of air passed into the lung, and on the stimulus ceasing the lung dilated to its normal extent. The cat's lungs could not be induced to constrict like that; vagal broncho-constriction only occurred readily in dogs and rabbits. The reason was that in cats there were well marked broncho-dilator nerves as well as broncho-constrictors. If the

bronchioles of a cat were made to contract by means of such a drug as pilocarpine, and if in this condition the vagus nerve were excited, it was found that the amount of air going into the lungs rapidly increased. When the stimulus ceased there was constriction again back to the former condition. There was reason to believe that other animals also had broncho-dilators, but they were not so well developed as in the case of the cat. The fact that when the vagus was excited the bronchioles became constricted was of no direct significance in relation to asthma, unless that constriction could be produced reflexly. Such a constriction had been obtained by stimulation of a number of nerves, but particularly the fifth nerve-endings on the septum of the nose at a spot high up and well back. If the medulla were under the influence of anæsthetics, no reflexes of that kind could be obtained. To show the effect, the animal had its cerebral hemispheres destroyed, but its medulla intact; on now exciting the nose with an electric current the bronchioles gradually became constricted, the constriction being very prolonged, and of a permanent character. Reflexes from the stomach of a similar type had been obtained.

Pilocarpine or colchicine excite the nerve-endings of the vagus and in that way cause some effect on the heart-beat with broncho-constriction coming on often in a series of waves, until there may be complete constriction of certain bronchioles. Tracings were shown from the same animal after injections of pilocarpine in which a dose of atropine was given; this immediately produced dilatation of the bronchioles, apparently by paralysing the vagal endings. Other tracings were exhibited showing the effect of lobelia, which seemed to have an action identical with that of nicotine. There was broncho-dilatation, but not of so permanent a character as that caused by atropine and its allies. Other slides illustrated the over-distension, and it was stated that no theory of asthma could be complete which did not account for the over-distension of the lungs. The act of inspiration was practically a suction force, and was a powerful one, much more so than the force of expiration, which was derived largely from the elastic tissue of the lungs; so that if broncho-constriction occurred, air might be sucked through the constriction by reason of the force brought into play, but the elastic recoil of the lung would not be sufficiently powerful to expel the air through the constricted area rapidly enough, and so another inspiration would take place before complete expiration. In this way, over-distension of the lungs would ensue. It was important to realize that over-distension of the lungs diminished the force of expiration—*i.e.*, the more the lungs were distended the feebler would be the power of expelling air afterwards. If one lung were dilated to its full extent, and egress of air through the trachea were prevented, one would expect that this lung would empty itself into the other lung. But that did not happen. On the contrary, a lung which was only a quarter-full emptied itself into a lung which was nearly fully dilated. This could be proved by mathematics, but he illustrated it by two rubber balls with air-tight connexions to the prongs of a Y tube; when either ball was blown up, it showed no tendency to empty itself into its fellow; so that in an attack of asthma as a result of broncho-constriction, a vicious circle was established; the more the chest expanded and

the lungs over-distended the weaker its powers of expiration became. Another factor to remember was the CO_2 . By giving an animal CO_2 one constricted its bronchioles; and roughly, within certain limits, the more CO_2 in the circulation, the greater the tendency to broncho-constriction. Some authorities had suggested that the sputum of the asthmatic might contain a toxin, and others that the Charcot-Leyden crystals might be a factor in the attack. He had tested these points and found that neither the sputum nor solutions of the crystals had any effect when injected into animals. There were two main hypotheses as to the causation of asthma, but he thought the vascular one had been largely given up, because no condition of the mucous membrane of the bronchioles was known, unlike that of the nose, in which great vascular turgescence could suddenly occur. The original hypothesis seemed to be based on the existence of a semi-erectile tissue in the mucous membrane of the nose, which could at a moment's notice become engorged, and it was argued, therefore, that the mucous membrane in the bronchioles, by becoming turgescient and swelling up, might cause a temporary block. But there is no such mucous membrane as this in the bronchioles, and such a block could not be produced. Furthermore, if such a mucous membrane obtained, then drugs like nitrite of amyl and ether, which caused irritation of the bronchioles and vascular engorgement, would be the worst things to give during an asthmatic attack. But amyl nitrite, taken by inhalation, would act immediately on the mucous membrane of the bronchioles and would dilate the local vessels before any others in the body; it would thus be the worst possible drug to give, but amyl nitrite exerted a beneficial action in these conditions. Thus in treatment three things had to be considered. First, the requisite hygienic measures must receive attention—food, locality, &c. Then the medullary neurosis must be treated, for there must be that predisposition. Recently, at Cambridge, a patient found that he invariably got attacks of asthma when he slept in one particular room, but that he did not when he slept in a similar room on the other side of the street. There was no difference in the hygienic conditions of the two houses, yet he was convinced he would get an attack in one house and not in the other, and so it happened. Suggestion must enter largely into the question. Thirdly, the exciting cause should be treated by one of the methods which Dr. Wall had described.

Dr. HERTZ said that, as a sufferer from asthma, he could give some personal details which might be of interest. He started having asthma when aged 13, and it was a long time before he realized that there was a mental side to it. For years he had an attack of asthma before every examination he went in for, in spite of the fact that at Oxford, except at examinations, he did not have asthma. It had been his custom to take arsenic and iodide for several days before an examination, and bring some powder to inhale at night during the period of the examination. But having fully realized the mental factor, he, on going up for his final M.B., made up his mind to have no attack, so he made none of his usual preparations. He did not have an attack, and it was the first time he had gone in for an examination without one. The next most important factor was the atmosphere, because at some places, whatever amount of will was

brought to bear, an attack came on, though its severity might be reduced by psycho-therapeutics. Recently, when he had to be in a place where he knew he would get an attack, he took a book to bed with him; when he awoke and had an attack, if the book was either an exciting novel or one which required much mental concentration, he was able to cut short the attack. The few patients he had been able to persuade to do likewise had received similar benefit. He agreed that suggestion might enter into the supposed association of certain houses or towns with asthma, but many times, when going to new places, without having any idea of asthma, he had had an attack. For instance, last year he toured about 3,000 miles in his motor, visiting many parts of England he had not been to previously. Such a place was Salisbury, where he had a severe attack, but as soon as he got clear of that city the asthma went, and he had no more of it until, on his way back from Land's End, he re-entered Salisbury. It seemed as if there was something in the atmosphere of Salisbury which affected him, though it might not affect other people similarly. The only true cure for asthma was not to have it. That could be done by avoiding residence in the places which caused it. If children had it at home, it was important to send them to school at a place where they did not have it. By avoiding attacks for a series of years one accustomed one's nervous system to being without attacks, and that had been so in his case, for he had been almost free from asthma for some years. In his own case the condition of the stomach had also an enormous influence. When in an asthmatic neighbourhood, if one starved oneself after six o'clock, that would greatly diminish the asthma.

Dr. E. I. SPRIGGS said the Section was fortunate in having heard Dr. Dixon's clear account of the experimental aspect of the subject; the work of Dixon and Brodie on asthma was a credit to British physiology and of practical value to medicine. Dr. Hertz's remarks reminded him of the admirable account of the disease written by Steavenson, who chose the subject for his thesis. But in his case the asthma came on after the examination; while the actual strain was on, he got along fairly well. Starvation, as mentioned by Dr. Hertz, was an important point. For instance, a subject of asthma, who was travelling on the Continent, and after an exceptionally long journey arrived at the hotel at 9 o'clock at night, very hungry, and, contrary to custom, dined heartily at that hour instead of at 7, precipitated an attack of asthma which recurred each evening for several days. He had notes of thirty-nine cases of asthma which he had treated, and odd things occurred in them, showing how important it was to study the individual patient. One patient's attacks began with a tertiary syphilitic rupial eruption. One hospital patient said he got attacks on Mondays. He usually took 12 pints of beer on Friday, 8 or 9 on Saturday, and none on Sunday. He gave up his beer, and was seen for two months, during which time he had no further attacks of asthma. Malaria was a well-known antecedent to asthma, and he had recently seen an example of this. One could often learn of inheritance in the condition; in his thirty-nine cases there was direct transmission in thirteen, and neurosis in the family in another eight. The treatment could be divided

into the treatment of the patient and that of the attack. Hygienic measures he regarded as of the greatest possible importance; the nose condition, constipation, fatigue were all important, and by looking to those things many people could be kept free of asthma for a long time. One gentleman had had asthma every winter for five years, but during the last winter he did not have it. He had not previously known the importance of not eating late at night nor over-tiring himself, nor yet allowing his bowels to become constipated. He had known constipation to be directly followed by asthma. Most physicians said they had seen a few cases in which treatment of the nose condition had cured the asthma; he had seen two cases in which the nasal operation made the asthma worse. One patient was plunged into a deplorable condition by having polypi removed. Still, there was undoubted benefit from treating any abnormal nasal condition in so many cases that it should always be done. It was important, in speaking of cure, to make sure that the disease was genuine asthma, and not a neurotic condition simulating it. He described a case in which the disease was closely simulated; but at every third or fourth breath the chest filled and relaxed with absolute freedom. On examining the lungs he found there was a spasm produced in the upper air-passages, and in the intervals of this the air could be heard to enter and leave the lung with perfect freedom. There was hypertrophy of the mucous membrane over the turbinate bones, and this was treated surgically with good results. In the literature he found 737 operations on the nose, and 324 were reported cured. The duration of the cure was very important, because all were familiar with post-operative quiescence. He had seen respiratory exercises do much good in asthma, combined with the Danish motions. Good results had been reported from the use of the pneumatic chamber at Brompton Hospital, in which pressure could be regulated. He would like to hear from Dr. Wall whether it was still being employed. Judging from his own cases, he believed the hope of successful treatment varied according to the nature of the case. If there was freedom from asthma between the attacks, and the patient was healthy, treatment could be undertaken boldly, with the feeling that good would result. But if there were any remains of bronchitis at the bases of the lungs treatment was more difficult. Tucker's spray was largely used by people who had got into that chronic condition; he knew two such, and they were snuffing at the spray all day. He had not found either Tucker's spray or Oppenheimer's very useful in acute attacks. He usually gave a prescription containing iodide of potassium, lobelia, and belladonna, and it did keep off attacks in a number of patients. He knew it was more in accordance with the ideals of the Section to give those drugs separately, and note their effects; but having tried that, he had found none of them separately give the same effect as the combination. In attacks, besides chloroform and morphia he recommended the injection of $\frac{1}{100}$ gr. of atropine, repeated in a couple of hours, or in twenty minutes, if the case was a severe one. Amyl nitrite was good if the patient was tired, and could drop to sleep in the short interval of freedom it produced and remain so. Adrenalin was used largely, and he had found an interesting case recorded

by Haplan and Brooks. One woman had had 2,000 injections in three years. She ultimately died, and the arteries were found thickened, with vascular and necrosed patches. Still, it did not follow that a reasonable therapeutic use of the drug was harmful.

Dr. CARMALT-JONES said there was a form of spasmodic dyspnoea which occurred frequently in chronic bronchitis, and he desired to say a word about the treatment of this variety of asthma. Fifteen months ago, in Sir Almroth Wright's laboratory, he investigated the bacteriology of chronic bronchitis, and from a patient he isolated an organism. He found the patient's opsonic index was low to that organism. He offered to try inoculation for her, and she consented. Two days later she came and said that though her cough was no better, her asthma was remarkably better, and she got up and lit the fire for the first time in seven weeks. He had collected notes of fifty-four cases of chronic bronchitis, all suffering from some degree of spasmodic dyspnoea, whom he had treated with the same vaccine, and of those, forty-one had shown some improvement. In some cases there was a complete cessation of dyspnoea for various periods, one of them up to six months, in people who had been the subjects of severe dyspnoea. In others there was simply a diminution in the frequency and severity of the attacks. If his patients left off the inoculations, they generally had a recurrence, but if they were inoculated once a fortnight, they remained much freer from attacks than before. Thirty-four had complained of difficulty in sleeping, and twenty-nine of them reported improvement in that direction. Thirty-eight complained of shortness of breath and difficulty in carrying on their work, and thirty-four had improved in that respect. He did not think any of them had been completely cured. The inoculations seemed to have no effect on the cough, merely on the dyspnoea. Other people, working on similar lines, seemed to have cured the cough, and when that was so, the dyspnoea was also improved. He thought the asthma in chronic bronchitis was a special toxic phenomenon, which occurred only in persons suffering from catarrh. The organism he found was one which he did not find described in books; it resembled Friedländer's, but differed in its fermentation of sugars. It was a polar-staining bacillus; it was negative to Gram, and did not liquefy gelatine, or ferment sugars. He believed it had a definite bearing on a large number of cases of bronchial asthma.

Dr. GRAY DUNCANSON said that anything which contributed to placing the treatment of asthma on a scientific basis was most acceptable to the profession. In general practice one saw many cases of asthma, and how frequently they passed into the hands of the quack and charlatan. A point not included in the paper, but which Dr. Spriggs alluded to, though he did not elaborate, was the advantage of physical and breathing exercises in the treatment of asthma—in those cases of spasmodic asthma, the origin of which could not be traced. Occurring during adolescence, breathing exercises were very beneficial; in carrying them out it was most important to attend to certain details—inspiration and expiration should be of exactly the same length of time, accurately measured, the

intervals being half that duration. In the treatment of acute attacks of spasmodic asthma he had found nothing so efficacious as a small dose of morphia, combined with atropine, hypodermically. It should be given by the physician and only at the height of the attack. He had used it for years, and had never seen the drug habit follow, and no one was surely a better judge of that danger than the general practitioner.

Dr. WALL, in reply, said he thought the Section might congratulate itself on the many points which had been brought out in the discussion. It was gratifying to hear Dr. Dixon's remarks on the experimental work on the subject, and in anticipation of Dr. Dixon's presence he (Dr. Wall) only lightly referred to that aspect; there could be no doubt that Dr. Dixon's paper was one of the most important which had been contributed to the literature of spasmodic asthma. He had himself noticed that it was difficult to distinguish the difference of the results produced by lobelia and nicotine respectively. He believed the result in both cases was due to the nauseating effect, though he did not know to what that effect was due. With the tendency to vomiting there seemed to be a relaxation of the unstriated muscle, in which the bronchial tubes partook. Lobelia had been retained in the *Pharmacopœia*, but tobacco had been eliminated. He believed the experience of asthmatics would favour the retention of tobacco and the elimination of lobelia. The remarks concerning the Charcot-Leyden crystals were very interesting. He (Dr. Wall) admitted that asthma was often associated with neuroses, but thought the psychical element was apt to be overestimated, as there was scarcely a chronic disease which had not some neurotic association. Association of a disease with a neurosis did not necessarily mean it was the result; it might be the cause. It was easy to understand an asthmatic becoming neurotic, as the recurrent attacks threw a great strain on the nervous system. Moreover, the frequent association of asthma with nasal trouble made it difficult to accept neurosis as the constant cause of asthma. The power of inhibition of attacks was well known to all who had studied the subject. The most remarkable instance in literature that he could remember was that of a patient subject to spasmodic asthma, whose daughter was subject to hysteria. The patient was one day having an attack of asthma when the daughter, who was in the room, had a hysterical fit. The asthmatic patient was excited about the hysterical fit, and ran downstairs to get a glass of water, without any dyspnoea. Having administered the water, and the daughter having recovered, he sat down and resumed his asthma. But it did not follow that in that father's case the asthma was a pure neurosis. Was it not conceivable that under the influence of the great emotion his brain put forth inhibitory impulses, which, in other circumstances, were not operative? It was admitted that much could be done for asthma by residence in suitable localities, but he had urged that that could not be considered an ideal mode of treatment. The desire was to cure the patient, though that was seldom possible. Dr. Spriggs's case concerning the weekly beer was very interesting, and Dr. Salter quoted a similar case—a Sunday morning asthma due to eating cheese on Saturday night. In

his own series of eighty-nine cases he had several parallel cases. He had not had much experience with regard to respiratory exercises for the condition; in the few cases in which he had seen them applied there was no marked benefit. He believed the pneumatic chamber had been abandoned in the treatment of asthma; the chances of improvement did not seem great enough to make it worth while risking a severe attack, which was a common result. Hysterical pseudo-asthma was well known, and it was not infrequently confused with true asthma. One had to be very careful in the case of women between 45 and 50 who complained of asthma following meals. Those attacks were probably cardiac in origin, and one could often find other hysterical manifestations in the patient. He knew very little about the bacteriology of chronic bronchitis. Any further experiments on those lines would be welcomed, and if, as he did not anticipate, it was found that spasmodic asthma was causally connected with a particular micro-organism, and could be cured or relieved by inoculation, everyone would be delighted.

Therapeutical and Pharmacological Section.

April 6, 1909.

Dr. T. E. BURTON BROWN, C.I.E., President of the Section, in the Chair.

Experiments and Experiences, Pharmacological and Clinical, with Digitalis, Squill, and Strophanthus.

By J. GORDON SHARP, M.D.

BIO-CHEMICAL STANDARDIZATION.

Dr. W. E. DIXON and Dr. G. S. Haynes read a paper before the Therapeutical Society on November 25, 1905, entitled "Bio-Chemical Standardization of Drugs." This paper was published in the *Pharmaceutical Journal*,¹ so that it was directly brought before the pharmacists, and it has done good, for pharmacists have come to insist upon having from the manufacturers a guarantee that preparations which cannot be chemically standardized should be tested pharmacologically. Dixon and Haynes suggested that the minimum lethal dose for a 20 gm. frog should be 2.5 m of either tincture of digitalis or tincture of squill, and for a 17 gm. frog should be 0.25 m of tincture of strophanthus, and that the heart should stop in sixty-six minutes in the case of digitalis, in one hundred minutes with squill, and in forty-eight minutes with strophanthus. These quantities are convenient to employ in actual practice, for a male frog weighs from 17 gm. to 20 gm., although, of course, specimens are often met with which weigh as high as 35 gm. or 39 gm. Seeing that one wishes to know the dose which will stop the heart, it is not absolutely necessary that intact frogs be employed—pithed frogs may be used, provided that every care is taken in pithing the animals whereby bleeding is prevented. In the pithed animal Dixon and Haynes's figures work out as stated by these authors, who, however,

¹ *Pharm. Journ.*, Lond., 1905, lxxv, p. 754.

worked with the unpithed frog. Considerable experience has taught me that while tincture of strophanthus almost always stops the heart within an hour, good specimens of tinctures of squill and digitalis may not stop the organ till two hours have elapsed. It has long been known that digitalis would stop the heart of a frog when injected into the dorsal lymph sac, but the knowledge did not get beyond the pharmacologist's laboratory till Gottlieb showed that this was the only reliable method of standardizing the drug. The Americans were the first to see the practical side of this knowledge and to make use of it. Cushny and Barger and Shaw showed the advantages of this method of ascertaining the activity of certain drugs, and the last two named authors read a paper at the British Pharmaceutical Conference of 1904, giving their experiences with tincture of digitalis. Their paper paved the way for the publication of Dixon and Haynes already referred to, which converted the rank and file, so that now all wholesale druggists of repute have their tinctures of digitalis, squill, and strophanthus tested physiologically.

During the past four years I have examined many specimens of all three tinctures, purchased in the open market, and I have had several specimens which acted with no more potency than a like volume of tap water. In one charitable institution the tincture of digitalis supplied by contract was found to be practically inert; the stock was returned, but the new supply was equally inert, for 2.5 m, diluted to 5 m, injected into the dorsal lymph sac of a 20 gm. frog (pithed) had no effect on the heart, and this organ was beating vigorously at the end of eleven hours. This was confirmed clinically and independently, for the resident medical officers, who were unaware of my experiments, made complaints about the preparations and said that they had had no effects at all from teaspoonful doses. This circumstance will serve to show how necessary it is that all institutions should have the preparations supplied to them examined by an independent authority.

(I) DIGITALIS.

The whole subject of active preparations is a medical one with a pharmaceutical side, and one wishes that medical men themselves would take a more lively interest in it.

Causes of Inert Preparations Occurring.—On talking over the subject of the potency of tincture of digitalis with Mr. Lancaster, pharmacist to the Leeds Public Dispensary, he suggested that we might investigate

some of the points which may lead to inert preparations being placed on the market. Our work has led to some interesting results: (1) It is doubtless a fact that leaves other than those of *Digitalis purpurea* are often gathered by collectors; (2) leaves are evidently often allowed to steam before they are thoroughly dried, and the ferment contained in them acts on the glucosides and decomposes the latter.

Test for Activity of Leaves.—I suggested a rough test for the presence of the ferment in dried leaves. The test is based on the presumption that if the leaves have been carefully dried the ferment would be present in abundance; contrariwise, if the leaves were allowed to steam, then the ferment would itself have been destroyed. I know it is open to many objections, but nothing more is claimed for it than that it is a rough test. An infusion is made from the leaves to be examined, and 20 gr. of amygdalin added to it in the unstrained state; it is set aside in a wide-mouthed stoppered bottle and kept at a temperature of 30° C. (86° F.) for five hours; if plenty of ferment be present in the leaves the preparation develops an odour of bitter almonds, and if a glass cover with a drop of nitrate of silver solution be laid over the mouth of the bottle the silver solution will in a few minutes become milky, due to the hydrocyanic acid resulting from the splitting up of the amygdalin by the ferment turning the silver nitrate into cyanide of silver.

Time for Collecting.—Mr. Lancaster and I collected or had collected for us in the neighbourhood of Leeds six batches of leaves. These we examined botanically, and we then dried them at a temperature of 21° C., or 70° F. Leaves of first year's plants and second year's plants, both those which had and had not flowered, were collected, and no distinction was made between large and small leaves. We made collections on August 11, September 4, September 11, September 19, October 10, and October 22, and, contrary to the usually accepted view, we found that the leaves collected on October 22 were quite as active as those collected in August.

How long do the Dried Leaves retain their Activity.—Mr. E. M. Holmes, curator of the Pharmaceutical Society's Museum in Bloomsbury Square, botanical referee to the British Pharmacopœia Committee, and one of the greatest authorities on the botanical source of drugs, was kind enough to furnish me with a small quantity of leaves which had been in the Society's Museum for eight years, dryness being insured by quick-lime placed in the hollow stopper of the bottle in which the leaves were contained. Mr. Lancaster made a tincture from this specimen, and I examined it on three occasions in the frog and found it up to the highest

standard of toxicity. I also examined it clinically, but of this I shall speak later. Fortune favoured us still further. In the course of my removal to another house I discovered lying on the top of a cupboard in my laboratory a parcel of digitalis leaves carefully tied up in several papers. These had been bought in the open market eleven years before. A tincture was made from them, and on pharmacological and clinical examination they were found to be of high average toxicity. The essential thing, then, it appears is that the leaves should be kept free from damp.

How long does a Tincture of Digitalis retain its Activity?—I have on several occasions examined tinctures which were respectively twelve, thirteen, and fourteen months old, and always found them toxic. In the last eighteen months I had a standard tincture prepared, and this I examined periodically every two and a half months. At the end of thirteen months it was quite active to all tests, but at the end of fifteen months it failed to stop the heart of a 37.5 grm. frog in suitable doses. Therefore one may conclude that tincture of digitalis begins to markedly deteriorate in activity between the thirteenth and the fifteenth month. To be well within the mark, one might say no tincture should be employed which is over twelve months old. Other points were investigated, but as they were more of pharmaceutical than of purely medical interest Mr. Lancaster will publish them in another journal.

Relative Toxicity of the Three Tinctures.—It is stated by some authorities that, volume for volume, tincture of strophanthus is nine to ten times more toxic than the tinctures of squill and digitalis. With this I am not in agreement, for strophanthus is one of those drugs which, like curare, is relatively more toxic when injected under the skin than when it is given by the mouth. If injected under the skin the dose should be relatively smaller, but when given by the mouth, as it almost always is given, I find that even as high as 30 m of tincture of strophanthus can be given and repeated every four hours for forty-eight hours without any untoward result.

Action of Digitalis on the Frog's Heart.—Here I only intend to speak of certain outstanding points which seem to me worthy of special note. Digitalis stops the heart less surely than, and not so suddenly as, strophanthus. The venous sinus and auricles may be beating while the ventricle is still in diastole, or it only beats very occasionally. Pinching may cause it to beat four to eight times, and it cannot again be made to beat till some time has elapsed. Finally, no amount of pinching will elicit a single contraction. Systole is never so extreme, and it takes

longer time to develop than with strophanthus. Is it true systole, or only molecular tension, whereby the muscle-fibres tend to occupy the smallest volume? As in the case of stoppage after strophanthus, the ventricle gets smaller and smaller without the appearance of contraction.

Pharmacological Action in Man.—Here again I only intend to speak of points which seem to me worthy of mention. When fairly large doses of the tincture—20 m to 30 m—are given, vomiting may take place, but nausea is much less than with like doses of tincture of squill, and diarrhoea is seldom present. (This, however, occurs after larger doses than those mentioned.) It has no irritant action on the kidney, and its successful exhibition is always attended by a large flow of urine. Its diuretic action is indirect, or, in other words, it acts by bringing more blood to the kidneys. The vomiting is not due to a local irritant action on the gastric mucous membrane, but is due to a poisoning of the tissues, for it occurs equally after pills coated with keratin have been given, whereby all action on the stomach is avoided. Of course, there is a local action on the gastric mucous membrane, and in some patients this membrane is so easily affected that the smallest doses cannot be tolerated in liquid form, but this is a different thing. The vomiting often lasts thirty-six hours after all digitalis has been stopped.

Recovery of Digitalis from the Urine and Stomach Contents.—When digitalis is given in ordinary doses it is excreted by the ordinary channels. When large doses are given, and more especially if they are continued over a long period, the tissues may become supersaturated with the drug, the ordinary channels are unable to cope with it, and the digitalis is, in addition, excreted by the stomach. Can it be recovered from the urine and stomach contents? I have evaporated at ordinary temperature several 40 oz. quantities of urine from patients taking full doses of digitalis. The dry extract was treated with chloroform containing 2 per cent. of absolute alcohol. The resultant of this treatment gave no reaction with phenylhydrazin till a portion of it had been boiled with strong hydrochloric acid, showing that probably one had to deal with a glucoside. A portion of this chloroform and alcohol residue gave with strong sulphuric acid a red-brown reaction which bromine and heat turned to violet. However, the most conclusive test was obtained when a portion of the residue was rubbed up with chemically pure sand and normal saline filtered and perfused through the blood-vessels of a frog. The blood-vessels were first perfused with normal saline till a constant volume was being passed in a given time, when the saline containing the supposed poison was turned on. Bit by bit the flow lessened till it

almost ceased. Normal saline containing a trace of sodium nitrite was next turned on, and the blood-vessels gradually relaxed and the flow returned to normal. The vomited material (free from all admixture of fresh ingested digitalis, and at the same time quite free from colour) was treated in the same manner as the urine and with a like result. I am still working at this subject, but it is a very long and wearisome experiment. Yet in time I hope to be able to obtain more definite results one way or other. One has to wait for appropriate cases to crop up, because mild poisoning cases are not met with every day. Recently I have somewhat simplified the process, and this will hasten the work.

Antagonism of Digitalis and the Nitrites.—In 1892 I was called to see a patient who had been taking tincture of digitalis and having the medicine repeated again and again without his doctor's knowledge; the result was not sickness or vomiting, but a marked irregularity and intermittency of the pulse. The digitalis was, it is needless to say, at once stopped, and he was prescribed sodium nitrite. The irregularity and intermittency at once stopped. Of course, the stoppage of the cause may have had a little to do with the obliteration of the intermittency and the irregularity, but this circumstance occurred so quickly that one must attribute it in great measure to the action of the sodium nitrite on the peripheral vessels. C. R. Marshall has shown that digitalis and the nitrites act so directly opposite to each other when they are perfused through the blood-vessels of a frog that if they be mixed together in exact proportions no action takes place, or, in other words, the contractor action of the digitalis so balances the relaxant effect of the nitrites that the vessels are unaffected. At first sight the natural deduction to be drawn from this experiment would be that sweet spirit of nitre and digitalis should not be prescribed in the same mixture. A little reflection will serve to show the fallacy of reasoning on these lines. In the experiment just mentioned the two bodies are applied directly to the tissues, and the action must be either to contract or relax. No other course is possible except when the proportions are so adjusted that the contraction balances the relaxation. When, however, the remedies are given by the mouth the two substances have to go the round of the circulation, and each takes its own special selective action and in its own time, and there is a "special time-action." The nitrite acts quickly, while the digitalis acts more slowly, so that the nitrite leaves the peripheral vessels (the seat of its action chiefly) in a condition especially suitable for the later action of the digitalis, and the heart is spared work in that it has not so much peripheral contraction to overcome.

Therapeutics.—I now come to the last and most important section, for all our pharmacology, physiology and pathology are vain unless they contribute to the relief of suffering. Here let me add in parenthesis that we are sometimes, perhaps too often, given to investigate the poisonous side of a drug rather than its therapeutic side. This unfortunate circumstance has often led to drugs and chemicals being for years left on the shelf of the laboratory when they should have been on the dispenser's counter. Witness the case of cocain. Pharmacology does no more than point the way, and this is all the seeker after truth will claim for it. It is a pleasure to come to treat of such a valuable drug as digitalis, and if I might dogmatize I should say that there is one and only one real heart tonic—viz., digitalis, and that if it fails nothing else will succeed. In introducing it to the practice of medicine Withering has proved to be one of the greatest benefactors of the human race. Given a patient with distended right heart, pulmonary congestion, low tension pulse and general distress, all manifestly due to a cardiac lesion, digitalis judiciously exhibited gives relief to the patient, encouragement to the practitioner, and makes him feel that there is something in his art of which he has reason to be proud.

Proofs of the Title "Heart Tonic."—One may here ask and try to answer the questions: (1) What is the true test of this or that drug being a cardiac tonic? (2) How can one demonstrate that digitalis is the best heart tonic we possess? One must rely on indirect rather than on direct evidence. To say that this or that drug is a heart tonic because it has toned up the pulse, relieved the palpitation or lessened the area of heart dullness is unreliable evidence, for there is room for so much divergence of opinion on these points. (1) But if a patient has the train of symptoms previously described (pulmonary congestion, dyspnoea, dropsy of the lower limbs, and so on), and he is sent to bed and carefully nursed and he gets well, one may safely conclude that all he required was physiological rest. This happens over and over again; one may go further and say that the cases which do not respond to this line of treatment are comparatively few in number. Now suppose that while this patient was undergoing this rest treatment he was at the same time given one of the reputed heart tonics, the latter would have undeservedly received the credit. This is the reason why from time to time drugs have been classed as heart tonics without the shadow of a title to the claim. If, on the other hand, rest has failed and a drug has succeeded there is presumptive evidence that the drug in question deserves the credit. I have had these points before me in carrying out my

experiments. (2) How, then, can one prove the superiority of digitalis? The answer is that it has succeeded after strophanthus and squill have been tried, proved and failed. The proof of both (1) and (2) will appear when I come to give short records of a few cases, but before I do this it will be convenient to discuss the virtues of the various preparations of digitalis usually employed.

Preparations of Digitalis.—(1) The infusion of to-day contains only a trifling amount of the glucosides, and is not of much service. The old Edinburgh infusion long retained a reputation for efficacy, and this may have been because it was in reality a weak tincture, for it contained spirit of cinnamon, and this was added to the hot water before the leaves were placed in to infuse. (2) The present-day tincture is the best preparation, and the most convenient to employ. It acts best when it is given in maximum doses and repeated every two or three hours, so that the tissues may be saturated as rapidly as possible. If it be given with 20 m to 30 m of the spirit of nitrous ether, the peripheral vessels are relaxed and the heart is spared work. Our forefathers employed the latter without knowing the reason why, but experience and experiment have proved the wisdom of their practice. Occasionally the tincture is rejected, and when this is the case it should be stopped, and if necessary the drug should be prescribed in pill form. (3) Baillie's, Abbot's, or Guy's pill, for it is known by all three names, is useful where the tincture or infusion is not tolerated. The blue pill is laxative; the squill is a valuable aid to the other ingredient, digitalis. Often I prescribe one every two or three hours till twelve or fifteen have been taken. (4) Occasionally the mercury salivates by the time six pills have been taken, and I prescribe a pill containing 1 gr. of dry digitalis leaves, 2 gr. of dry squill, and 1 gr. of dry extract of cascara sagrada. It is useful where constipation is present, and I have seen a dropsy disappear in an extraordinarily short period of time when it has been given. Both (3) and (4) belong to compounds of digitalis, but it is here convenient to mention them. (5) The glucosides of all makers I have tried—crystalline and non-crystalline—but I have never been satisfied with them, and I have never known them to succeed when the tincture and compound pills have failed.

How long does Digitalis take to act.—The general belief is that the drug takes thirty-six to forty-eight hours to show any appreciable action. The fact is, digitalis begins inappreciably to act as soon as it is given, but if it is given in minute doses so that the tissues are able to deal with it and to excrete it almost as fast as it is ingested, then it may

appear to act very slowly ; but where it is given in the doses and repeated with the frequency with which I have indicated, it acts as rapidly as is desirable.

The following records will, I trust, support the various assertions I have made in the foregoing part of my essay.

Case I.—A woman, aged 68, who was suffering from cardiac dropsy, with the passage of 10 to 13 fl. oz. of urine in twenty-four hours and unrelieved by rest in bed, was given 20 m of the tincture every four hours, and at the end of eighteen hours she had taken 80 m, when the dyspnœa was relieved and diuresis was well established.

Case II.—Patients with a very low tension pulse are readily saturated with digitalis, hence diuresis is early established, as witness the following : A man, aged 52, who had been suffering from dropsy of the lower limbs and ascites depending on mitral disease, was confined to bed for a week before I saw him, so that he was in the right condition for the trial of digitalis. His pulse was soft, as already hinted, and he was passing from six to ten fluid ounces of urine in the twenty-four hours. I prescribed 15 m of tincture of digitalis every three hours. The first dose was given at 9 p.m., and by 5 next morning, or eight hours later, he passed at one time 10 fl. oz. of urine, and by 11 in the morning he had passed altogether 30 fl. oz.

Case III : Experiments with Tincture made from eight-year-old Leaves.—This record will serve a double purpose, for it will tend to prove that digitalis leaves may retain their activity for eight years if they are kept dry. It will also serve to show the rapidity with which the drug acts. A boy, aged 13, whom I see at intervals, suffers from mitral regurgitation following rheumatism, and I only see him when rest in bed fails to remove the dropsy from which he periodically suffers. His mother knows the importance of rest, and when his feet begin to swell he is at once sent to bed. On the present occasion a week's rest had failed to remove the dropsy from the abdomen, although the feet had gone down under the treatment. He was breathless on the least exertion, the abdomen was full of fluid, but the fingers could be insinuated deep down and the congested and enlarged liver could be felt. At 5 p.m. he was given 10 m of the tincture under examination along with 15 m of spirit of nitrous ether, and this was repeated every two hours while awake. By 1 p.m. next day he had only taken 50 m, and yet the breathing was easier, the abdomen had gone down an inch in circumference, and he had passed a large quantity of urine. The tincture was continued, and by the time he had taken 3 fl. dr. the

abdomen was free from fluid and the boy was practically well. Here the drug had acted in twenty hours.

Case IV: Tincture from eleven-year-old Leaves.—This experiment was conducted with tincture made from leaves eleven years old. Like Case III, it serves a double purpose. A vigorous man, aged 80, showed signs of heart failure with breathlessness, pulmonary congestion and swelling in the feet, gradually creeping upwards, which rest failed to remove; indeed, the swelling gradually increased and reached the knee. He was prescribed 20 m every eight hours, and at the end of twenty-four hours the change was marvellous, for all the symptoms were relieved, and by the time he had taken 3 fl. dr. every symptom had disappeared.

Case V.—With a record of this case I close this part of the essay. A man, aged 72, and an old friend, suffers from mitral regurgitation. He knows all the precautions that are necessary to obviate a breakdown, and he knows the first line of treatment when this does happen. Occasionally he forgets and throws a heavy strain on the heart, with the inevitable result. On the occasion of which I wish to remark he had been in bed five days, but the condition progressed. When I saw him he was unable to carry on a conversation without having to stop to take breath. His feet, legs, and abdomen were swollen, and there was disturbance of the lesser circulation, with the usual symptoms. At 4 p.m. he began to take Baillie's pill every two hours. In twenty-four hours he had taken ten pills, with the result that the swelling in the feet and legs had gone down, the abdomen was less, the breathlessness had so much improved that he was now able to carry on a conversation without effort. The pills were stopped, and the patient recovered by rest in bed and nursing.

It is unnecessary to quote other cases, for they are all the same. If a patient has within him the elements of recovery digitalis will so act on the heart and circulatory system that it will remove the dropsy and bring about the recovery of the sufferer, and that in a period of time much shorter than is generally supposed. Digitalis has had a bad name for tardy action, and this tradition has been handed down from generation to generation and has been accepted without inquiry, until it has almost become an article of faith and a test of therapeutical orthodoxy. Long experience and close observation have confirmed me in the belief that digitalis acts as rapidly as most remedies we possess.

I have asserted that if digitalis fails in relieving the effects of a breakdown in compensation in cardiac disease nothing else will succeed.

The following goes a long way in support of this contention: A man, aged 64, is a sufferer from mitral regurgitation, with a suspicion of tricuspid regurgitation with the usual failures of compensation. Digitalis has often worked marvels, but as time goes on the lesions increase in degree and a period is reached when everything fails. Early in December, 1908, he had what appeared to be the ordinary dropsy, from which he had so often suffered, but digitalis failed to perform what it had so often done. True, it did relieve the dropsy a little, but it removed it in patches, and this is always a bad sign. On December 18 he was prescribed 20 m of a standard tincture of strophanthus every two hours, and in thirty-six hours the ascites was a little less, and so were the feet and legs, and the breathing was easier. The tincture was now only given in 20 m doses once a day until January 8, 1909—that is, the drug was continued for three weeks, when it was stopped, for it was evident that nothing more could be got out of it. Baillie's pill was now ordered three times a day, but soon it had to be stopped because of diarrhœa, and, besides, it had not lessened the dropsy nor improved the general condition. Tincture of squill was now prescribed in 20 m doses four times a day, but with no better results, and, besides, it produced nausea and vomiting, and it also had to be stopped. On January 13 he was prescribed fresh decoction of broom, with 20 gr. doses of acetate of potassium three times a day, and this was continued for a week with no success; the disease progressed. Citrate of caffein in 10 gr. doses, with the same of sodium salicylate, was next tried, four times daily, but without any beneficial action. On January 26 the abdomen was tapped and 3 pints of clear fluid removed, but only with partial success. On January 29 he was once again given tincture of digitalis in 15 m doses, three times a day, but, unfortunately, it failed to act. The dyspnœa increased, so I again gave 20 m doses of tincture of strophanthus every four hours. This was on February 2. It, as before, relieved the dyspnœa somewhat and induced a feeling of comfort and general well-being, but had no effect on the dropsy of the various parts. The man died quietly two days later—namely, on February 4, 1909.

(II) SQUILL.

In my experience commercial tincture of squill does not vary so much as the other two; still, the same care has to be exercised in ensuring that an active preparation is obtained.

Action on the Frog's Heart.—Squill appears to act on all the anatomical parts of the heart, and if the chest be opened in the course of an experiment when the drug is in full action the auricles and ventricle are found to be beating most vigorously, with a suspicion of a long pause at the end of diastole. Systole is not so extreme as is diastole. To all appearance the work performed by the heart-muscle is much increased—much more than with either digitalis or strophanthus. As far as I can judge the heart (auricles and ventricles) stops simultaneously, and it stops because it is quite exhausted by the powerful stimulant action of the squill on the muscle-fibres, either direct or indirect, through the nerve mechanism.

Pharmacological Action in Man.—Squill in large doses—say 20 m to 30 m of the tincture—produces nausea, sickness, and often diarrhœa, but it appears to have only a trifling action on the secretory tubules of the kidney, and there is little irritant action on the kidney. Its exhibition is followed by the passage of a moderate flow of urine in cases of cardiac dropsy, so that its diuretic action is chiefly indirect, and thus through the vascular system like digitalis.

Therapeutics.—Dr. G. S. Haynes, in writing of the pharmacology of squill in the *Bio-Chemical Journal*, vol. i, No. 2, says: "I should like to enter a plea for the more extended use of squill in place of digitalis in cases of heart failure; it acts more quickly, more effectively, and does not cause any more, if as much, gastro-intestinal derangement." With this expression of opinion I am only in partial agreement, and even then only conditionally. If it can be proved that squill acts within a few hours—say three or four—then I believe it may give a temporary fillip to the cardiac organ till digitalis has had time to act. In my opinion and experience it only acts up to a certain point and then fails entirely. Even although it may be given in the largest possible doses, only a limited amount of action can be got from it. It is doubtful if it will remove a dropsy if previous rest and hygienic treatment have failed to do so, and yet, as an adjunct to digitalis in cases of cardiac dropsy, there cannot be any question as to its service. Especially is it of service in combination with digitalis when there is much pulmonary trouble, either in the form of a simple congestion or a bronchitis. This points to its having a special selective action on the lesser circulation, and this in turn may supply the clue to its successful employment in cases of simple bronchitis with distended right heart.

(III) STROPHANTHUS.

As the result of our investigations Mr. Lancaster and I found that an active tincture of strophanthus could always be prepared if the crude drug was bought in pods, and, of course, if the seeds were examined chemically and the usual care employed in preparing the tincture.

Modified Sulphuric Acid Test for Strophanthin.—The green reaction with sulphuric acid is often difficult to obtain with the cut seed, especially if the acid be not up to the standard. The following is suggested as a handy and reliable modification of the official test. A seed is "cut up" and placed in a white porcelain dish with 5 m of dilute sulphuric acid. The dish is then whisked in the flame of a spirit lamp, care being taken to avoid charring, and as soon as the acid attains the requisite degree of concentration the green colour appears, provided the seeds be of the true species—*Strophanthus Kombé*, Oliver. The seeds of various species of strophanthus deprived of their awns are so much alike that every care has to be taken in obtaining the true drug. A great deal too much has been said about the difficulty in obtaining the true and, at the same time, active seeds of *Strophanthus Kombé*, Oliver, and whenever a specimen of tincture has not come up to the expectations of the clinician he has at once said that the preparation had either not been made from the true drug or it had been made from non-active seeds. It seldom has occurred to him to ask if he was not asking more from the drug than it had to give. Now that strophanthus is being almost universally examined physiologically, there should be no excuse for anyone employing other than the true drug. The importance of this is great, for in time the drug will be assigned its right place in the list of remedies. If a maker buys pods having the botanical characters of *Strophanthus Kombé*, and the contained seeds are in good condition and give the chemical reaction for strophanthin, and if a tincture from these seeds stop the heart in a given dose and in a given time, there can be little doubt of the genuineness of the specimen.

Action on the Frog's Heart.—Strophanthus more surely stops the heart, and stops it more suddenly, than digitalis or squill, and further it stops it, or appears to stop it, simultaneously in all its anatomical parts—sinus, auricles and ventricle. Further, often when the chest is opened, although the whole heart is still, the ventricle is not in complete systole; indeed, it may be in moderate diastole and dark in colour, as if filled with dark-coloured blood. Soon the dark-coloured area becomes less and less, leaving a patchy surface, but finally the

ventricle becomes pale but without a single contraction having taken place. Its surface is rough and shrunken in appearance.

Pharmacological Action in Man.—I intend here only to speak of certain points which seem worthy of special remark. When comparatively large doses of tincture of strophanthus are administered—say 20 m to 30 m, and repeated for thirty-six to forty-eight hours, at intervals of two to four hours—one finds an irritant action on the kidneys, with the passage of a small quantity of urine and frequent urination. Seldom is there sickness and diarrhœa with the doses named. The slight diuretic action thus appears to be a direct one, and due to a stimulant action on the secretory tubules of the kidney. In cases of dropsy one never finds the large flow of urine which so often follows the administration of digitalis.

Therapeutics.—The pharmacological evidence (as one views it in its action on the frog's heart and in the excised mammalian heart) in favour of strophanthus as a heart tonic appears to the most sceptical as conclusive as it can well be. Unfortunately this evidence is not borne out by clinical experience, and few clinicians of twenty years' experience can be found to pin their faith to the drug; and, indeed, many deny it the title of heart tonic at all. Certainly, judged by the same indirect test which one applies to digitalis, it fails. At the risk of repetition it is necessary here to again mention the test applied. Given a patient suffering from mitral regurgitation with breakdown in compensation and its consequences—distension of the right heart, pulmonary congestion, difficulty of breathing and dropsy of the lower limbs—will tincture or other preparation of strophanthus remove the distension of the right heart and the pulmonary congestion, and in turn the difficulty of breathing? Will it bring more blood to the kidneys, increase the quantity of urine passed, and so remove the dropsy of the lower limbs?—and that after rest in bed has failed? In my experience it will not. True, it may by its stimulant action on the kidneys cause a little more urine to be passed, but only in this way does it act, and its action in this direction is trifling. Cases of this kind, as mentioned in another part, have subsequently yielded to the administration of digitalis.

However, I am certain strophanthus must possess some curative action, for within the last few years I have prescribed 20 m to 30 m of a toxic tincture every two or three hours in dyspnœa with a labouring heart and dropsy, and it has done good. The patient has slept, the dyspnœa has been relieved, and the sufferer has felt more comfortable;

but the dropsy has not been removed, even although the drug has been continued for three to four days. Here, again, digitalis has entirely removed all the consequences of the breakdown in compensation. I am unable to offer any explanation of this beneficent action of strophanthus. A combination of digitalis and strophanthus, like that of digitalis and squill, might be of service.

I have not touched upon certain controversial points—such as whether strophanthus is simply a muscle poison, or how far digitalis acts as a stimulant to the cardiac muscle, and how far it acts on the vagus ends, and how far it increases the muscular tone, and how far it acts on the excito-motor apparatus of the heart. Nor have I touched on the question as to whether or not squill in its action differs in degree or in quality of action from digitalis. I have tried to seek truth rather than to follow tradition, and if I have made dogmatic statements I have at the same time endeavoured to prove them by actual evidence.

DISCUSSION.

The PRESIDENT (Dr. Burton Brown) expressed the thanks of the Section to the author for his interesting and important paper. He said that in an institution with which he was connected those drugs were being examined a good deal, and the man who examined them found that the digitalis leaves of the first year were equally strong with those of the second year. He did not know whether Dr. Sharp had found the same thing. He also found that the mode of preparing tincture of digitalis by percolation did not exhaust the leaves, and probably the old way in which tincture of digitalis was made by putting the leaves into a large bottle and adding the required amount of spirit, and then leaving it for weeks or months, produced a stronger tincture than that obtained at present. In the case of hyoscyamus the second year's leaves were much stronger than those of the first year; but that was not true of digitalis.

Mr. SOPER said the Section had much to thank Dr. Sharp for, but he confessed he felt somewhat disquieted when he heard of the very short life of tincture of digitalis. He asked Dr. Sharp whether, from his own observation, he had found that in some mysterious way after keeping the digitalis the digitalin—the active principle—had gone; or did he mean that the efficacy of the drug was much reduced in the old tincture compared with the new? He could go back many years, and he thought we were inclined to err on the side of isolation, *i.e.*, isolating certain properties of drugs which Nature had handed to us. He knew more about the infusion of digitalis and the power of digitalis,

and that those forms were nearly always efficacious. He was not prepared to say that a tincture did not do pretty much the same as a preparation of quinine from bark: it took something but left something else, and he was not prepared to say how far that something else was not the potent remedy which existed in the fresh infusion. The author had shown how beautifully the leaves kept for years and retained their green colour, though he did not know whether that green colour had anything to do with efficacy. In a close-stoppered bottle the powdered digitalis combined with a grain of pil. hydrarg. and a grain of powdered squill did more good than tincture of either squill or digitalis. He pinned his faith upon those as a cardiac tonic and diuretic.

Dr. JAMES MACKENZIE said that to a great extent he was convinced that the whole subject of the clinical administration of digitalis would have to be rewritten, and rewritten from the standpoint that digitalis reacted in the human subject according to the nature of the disease. One might take a great number of patients and give them digitalis until they were poisoned, causing sickness and diarrhoea, yet producing little or no effect on the heart, beyond slowing it a little. Digitalis might be given to another series of patients, and in twenty-four hours there might be a definite reaction. But that reaction did not depend upon the drug, but upon the condition of heart from which the patient suffered. He had been able to demonstrate with certainty the very distinctive actions which digitalis and strophanthus and squill had upon the heart if one first considered the nature of the lesion in a given case. One might have a patient with dilated right heart and dilated left heart; digitalis given produced a good result. The same quantity might be given to another patient with identical symptoms, but with no result. That meant that the physician had to investigate the nature of the lesion which was the cause of the dilatation. The same was true with regard to the rhythm of the heart. He had been able in many cases to take a patient and say with certainty that in that patient he would produce one form of arrhythmia, and in another patient another form. Those forms of arrhythmia were excellent for giving a clue as to the nature of the damage or change in the heart. In carrying out a series of observations he found that when a patient reacted to digitalis he also gave the same reaction to strophanthus if pushed far enough. He was very much struck in the case of a young man with mitral stenosis, who had what he called the nodal form of arrhythmia, in which the ventricle started the contraction and the auricle contracted with it. In certain cases of that kind he could play upon the heart, making it quick or slow, by either exhibiting or withholding digitalis. He asked that young man in one of his periodic breakdowns what his experience was. He said that he had had many attacks of failure of compensation, and he found that digitalis acted very rapidly, that strophanthus did the same, but it took a longer time to do so. He (Dr. Mackenzie) thought the time had come when pharmacologists, especially those who were dealing with the heart, should begin to understand the fact that before dogmatizing on the action of any drug, they should recognize the principle that in the human heart digitalis and other drugs acted according to the nature of the lesion.

Dr. CHARLES W. CHAPMAN desired to express his agreement with Dr. Mackenzie concerning the last point he mentioned: he believed there was no one drug which was more abused than digitalis. He used it very much, and also saw much of the results of its abuse. Much harm had been done by prescribing it in the wholesale rule-of-thumb way which some practitioners adopted just because there was "something wrong with the heart." The lack of a correct diagnosis was at the bottom of such treatment. A very able man said to him not long ago that it did not matter what was wrong with the heart, he gave them all digitalis. Such a man as that was beyond argument. Many patients who had only some reflex trouble were dosed with digitalis. And what was perhaps even worse was the modern practice of giving cardio-vascular remedies, such as nitro-glycerin, simply because there was a sharp pain in the cardiac region. One could not say too much for the combination of blue pill and squill, and it was very important in hospital practice among the poor, who could not always afford to lie up. Provided they kept a watch on their gums they could keep up daily doses of that for weeks without anything but good. In some of the modern preparations one drop equals a grain, and he thought they seemed to answer very well.

Professor CUSHNY, F.R.S., desired to add a historical note to the paper—namely, in regard to the pharmacological standardization of digitalis. Of course, pharmacologists had known for thirty years the method of testing digitalis on frogs' hearts; in fact, the different glucosides were tested by Williams by that means when Schmiedeberg isolated them. But no practical use was made of that test until 1894, when Dr. Houghton began working at the subject, and very soon described a method of testing the relative strength of different preparations of digitalis, &c., on the heart of the frog. It was introduced by him into pharmaceutical practice in 1895, a point he wished to emphasize. The process was taken up by Parke, Davis and Co., in Detroit, where Houghton had become one of the staff, and subsequently by a considerable number of American firms, and by 1900 some half dozen of those firms were testing the drugs in that way. Several years later, about 1898 or 1899, the use of this method was introduced into pharmaceutical practice into Germany by Gottlieb, who appears to have been ignorant of Houghton's work. About eight years after Houghton's first publication it was taken up in England and used by several pharmaceutical houses. He believed that recent work had really added very little of importance to Houghton's results.

Dr. SHARP, in reply, said he found the old tincture of digitalis had no effect at all; the heart was vigorously beating at the end of eleven hours from the injection of the usual dose. That was a tincture fifteen months old. Sometimes the infusion in large doses might do good, and he thought the powder should be given when the tincture could not be taken. But the tincture was very handy—an important point. He was always a little sceptical about the infusion acting. He did all he could to guard against error. Dr. Mackenzie was known to be a master on the subject, probably he knew more than anyone about it; yet he did not agree with him in all he said. He (Dr. Sharp) had one case of undoubted

heartblock, for which, of course, digitalis should not be given. The pulse was only 36, and when he got some improvement he had the patient skiagraphed by a physician who was well up in the subject, and who said the auricles were beating at the rate of 72, and the ventricles at the rate of 36. Yet that man certainly seemed to be benefited by small doses of digitalis. He could not say that tincture of strophanthus had the same action as digitalis. He had given strophanthus in 20 m to 30 m doses repeated every three hours for forty-eight hours, and apparently with no effect. Then he tried digitalis and it had removed the dropsy. That seemed a fairly good proof of the superiority of digitalis over other substances. As he said in his paper, it happened over and over again that all the patient required was physiological rest; indeed, the cases which required digitalis were comparatively few in number; so he had always tried what rest would do first. He thought it would be agreed from what he had said that he had not abused digitalis; he had tried everything else before giving it. He had been aware of what Professor Cushny said, and—a point he naturally would not mention himself—the Professor had himself done much in connexion with the subject of standardizing digitalis. It was well known that it took a long time for information to filter through to the rank and file in every country.

Therapeutical and Pharmacological Section.

May 4, 1909.

Dr. JOHN INCE, Vice-President of the Section, in the Chair.

The Results of a Year's Use of Vaccines in General Medicine.

By W. HALE WHITE, M.D., and J. W. EYRE, M.D.

WHEN your secretaries kindly asked us to bring before the Society some subject of therapeutic interest it struck us that it might lead to a useful exchange of opinion if we laid before you every case which we have conjointly treated with vaccines, excluding those treated with tuberculin. Our only reason for excluding these is that tuberculosis is usually such a slow disease that it is impossible to draw conclusions as to treatment until many months have elapsed. The following cases have all occurred since January 1, 1908. Those at present still under treatment are not included, but with that exception we give every case which we have jointly treated with vaccines, so that we may as far possible arrive at a correct view of its value. We have grouped the cases under the following heads: (A) Genito-urinary System; (B) Alimentary System; (C) Empyemata and Subdiaphragmatic Abscesses; and (D) Miscellaneous.

(A) GENITO-URINARY SYSTEM.

Case I.—The patient, a female, aged 49, married, had suffered from multiple arthritis for nine months. It had been regarded as osteo-arthritis. No benefit had followed any of many lines of treatment. The gonococcus was not isolated from the vaginal discharge, but the

index to it was 4·0; therefore she was treated with a stock trivalent gonococcal vaccine in doses of from 5,000,000 to 10,000,000 at intervals of seven days; three doses were given. The arthritis entirely subsided and she was discharged cured at the end of a month.

Case II.—The patient, a female, aged 30, married, had suffered from multiple arthritis for three weeks. The gonococcus was isolated from the urethra. The patient was treated with trivalent stock vaccine. She received three doses of 5,000,000, 5,000,000, and 7,500,000 respectively at intervals of six days. She was discharged cured, and when seen three weeks later she was quite well.

Case III.—The patient was a male, aged 36. First attack of gonorrhœa. Two months later arthritis followed, and when the patient was seen it had existed two months. Many joints in the body were affected. The gonococcus was isolated from the urethra, and the patient was vaccinated with a vaccine prepared from his own gonococcus. Four doses varying from 5,000,000 to 25,000,000 were given at intervals of six days. He was discharged cured, and when seen three weeks later was quite well.

Case IV.—A male, aged 26. First attack of gonorrhœa; severe, prolonged, followed by multiple arthritis four months after the gonorrhœa. On admission the arthritis had lasted for three weeks; the gonococcus was isolated from the urethra and a vaccine was prepared. Owing to the number of generations that elapsed before the gonococcus was isolated in a state of purity, larger doses than usual were employed. The patient received three doses of 100,000,000, 100,000,000, and 200,000,000 respectively at intervals of twelve days. The opsonic index varied during this period from 0·7 to 3·6. The arthritis entirely disappeared in five weeks and had not returned five weeks later.

Case V.—The patient, a female, aged 45, seen with Dr. C. Ewart and Dr. J. Mitchell Bruce, had five weeks before we first saw her begun to suffer from pains and swelling in many joints. Remedies for gout were tried but did not relieve. The ears and the bridge of the nose became swollen and double conjunctivitis supervened, followed at the end of four weeks by severe cystitis, with blood and pus in the urine and high temperature, and pain over the bladder and both kidneys. When first seen by us she was very ill with cystitis and double pyelitis, vomiting, rapid pulse, and high temperature. She was so ill that it was thought likely that she would die. *Bacillus coli communis* was isolated from the urine and a vaccine was prepared,

the index to the organism being 0.35. A 5,000,000 dose of the vaccine from her own *Bacillus coli communis* was followed by a rise of the index to 1.6. Four days later a dose of 30,000,000 was injected, followed after ten days by a dose of 250,000,000. Improvement began directly after the first injection. The patient made an uninterrupted recovery, and when we saw her a year later there had been no return of the trouble. The range of temperature is well shown on the accompanying chart. (See Chart 1.)

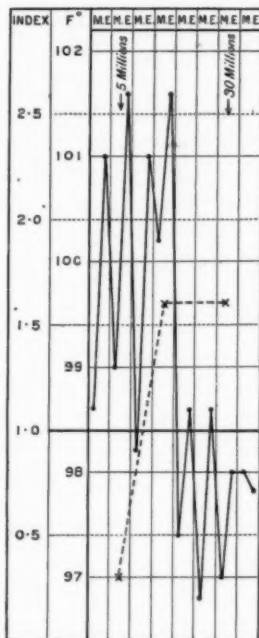


CHART 1 (Case V).

Bacillus coli infection. Temperature raised for five weeks before this chart begins.

(In all the charts the straight line indicates the temperature and the dotted line the opsonic index.)

Case VI.—The patient, aged 26, seen on March 18, 1909, with Inspector-General J. J. Dennis, R.N., had had enteric fever from Christmas, 1908, to the middle of February, 1909. From February 15 to February 25 the temperature had been normal; then high

evening and low morning temperatures suggestive of pus prevailed, and had continued ever since. On March 15 pus appeared in the urine, and the right kidney became tender. *Bacillus coli communis* and *Staphylococcus albus* were found in the urine, and vaccines of each were prepared. The index was 1·45 to *Bacillus coli communis* and 0·44 to the *Staphylococcus albus*. A dose of 5,000,000 of his own *Bacillus coli communis* vaccine was injected on March 20. By March 23 the temperature had become normal, and another dose of 5,000,000 was given, and a dose of *Staphylococcus albus* vaccine of 100,000,000. On March 29 there was still some pus in the urine and a dose of 100,000,000 *Staphylococcus albus* vaccine was given, and on March 31 a dose of 10,000,000 *Bacillus coli communis* vaccine was given. No further treatment was necessary, and the patient has remained quite well and has gained much weight.

Case VII.—The patient, a woman, aged 84, seen on July 6, 1908, with Dr. H. Sharman, had had a few weeks previously slight left hemiplegia, after which cystitis had slowly developed. *Bacillus coli communis* was isolated from the urine and a vaccine was prepared. Three doses of vaccine from her own *Bacillus coli communis* were given—viz., 2,000,000 on July 9, 5,000,000 on July 13, and 10,000,000 on July 20. No other treatment was adopted. By the 23rd she was well enough to get up and she made an uninterrupted recovery.

Case VIII.—The patient, an unmarried woman, aged 30, was admitted to hospital for pain over the right kidney region and severe cystitis of eight months' duration. *Bacillus coli communis* was isolated from the urine, a vaccine was prepared, and three doses of 5,000,000, 5,000,000, and 10,000,000 respectively were injected at intervals of six days. The pain subsided after the first injection, and the pus in the urine disappeared entirely after the third injection. The patient was discharged cured.

Case IX.—The patient, a woman aged 40, seen with Dr. H. W. Armstead, had been first seen by one of us in 1907 for left renal calculus. This had so destroyed the kidney that on consultation with Mr. A. A. Bowlby it was found necessary to remove the whole organ. The patient improved much after operation, but some time subsequently she began to suffer from recurrent attacks of pain in the right kidney, associated with pyrexia. She was seen on February 18, 1909, for these symptoms. There was no pus in the urine, but it contained large numbers of *Bacillus coli communis*, whilst the index to the organism was 0·6. She has been repeatedly vaccinated with large doses of a stock

vaccine and small doses of a vaccine from her own bacilli, but without any marked improvement. She refuses to be X-rayed, and will not entertain the idea of another operation.

Case X.—A man, aged 50, was admitted for severe aortic and mitral disease. He also suffered from retention of urine, due to stricture. For this catheters had been used, and he had cystitis. *Bacillus coli communis* was isolated from the urine. Two doses of 5,000,000 of vaccine prepared from this bacillus were given at intervals of seven days. There was no improvement in the cystitis, and he died from his heart disease six days after the last injection.

(B) ALIMENTARY SYSTEM.

Case XI.—The patient, a married woman, aged 38, seen with Dr. C. S. Pantin on March 18, 1908, had had, every three weeks for the last eight months, attacks lasting three days and consisting of nausea, heartburn, and a severe feeling of illness, with shivering and slight rise of temperature, which quite incapacitated her; during an attack she was always very constipated; since the attacks began she had lost much flesh. She had seen several medical men, and duodenal ulcer and atonic dilatation of the stomach had both been diagnosed at different times, but no benefit followed treatment for these conditions. It was found that washings of the stomach contained numbers of *Bacillus coli communis* and no other organisms. From her own *Bacillus coli communis* a vaccine was prepared. It was administered at intervals of about twelve days in doses gradually increasing from 5,000,000 to 50,000,000 until September 7, 1908, when she was seen again. Her weight had increased from 7 st. 9 lb. to 8 st. 10½ lb.; she looked so well that she was hardly recognizable; she had a good appetite and she felt well; her attacks were now very slight and infrequent. A washing from the stomach revealed the presence of *Bacillus coli communis*, but only in very small numbers. She was seen again on April 19, 1909; she then looked well, had maintained her weight, and had had no further attacks, but she suffered, as she had for many years, from some discomfort due to a right floating kidney.

Case XII.—The patient, a boy, aged 12, seen on October 17, 1908, with Dr. James Smith, had suffered from ulcerative colitis for four years, passing blood and mucus, and pyrexia, pain, and tenderness. No treatment had done any permanent good. *Bacillus coli communis* was isolated from the fæces. The patient was vaccinated with a vaccine

prepared from this; he had doses varying from 5,000,000 to 50,000,000 at average intervals of a week for three months. At the same time he was given two pints of soured milk prepared with lacto-bacilline and small doses of cyllin three times a day. The improvement was immediate and rapid. An examination on January 11 last showed that the atypical *Bacillus coli communis* previously found had disappeared from the fæces. The boy is now (May, 1909) perfectly well and has been so for many months.

Case XIII.—The patient, a male, aged 30, seen on November 6, 1907, with Dr. W. W. Shackleton, had had for five weeks severe diarrhoea with much blood. He was very blanched. He was treated with light diet, and astringent injections were thrown high up into the large bowel. He got much better, but in March, 1908, the old symptoms returned and continued until July 9, when he was seen again. An examination with the sigmoidoscope showed the whole mucous membrane to be intensely injected. A *Bacillus coli communis* was isolated from the fæces and a vaccine was prepared. A dose of 5,000,000 was given on July 18, 5,000,000 on July 21, 10,000,000 on July 31, and 5,000,000 on August 18. The patient then discontinued the treatment, his symptoms being improved, but they returned in October. Four doses were then given at fortnightly intervals, the patient again recovered, and when last heard of (May, 1909) was quite well.

Case XIV.—The patient, a male, aged 17, seen on May 16, 1908, with Sir William Allchin and Dr. E. H. Lendon, had had severe ulcerative colitis for many months, passing mucus and much blood. He was very ill and was confined to bed. *Bacillus coli communis* was isolated from the fæces and a vaccine was prepared. He was given small initial doses, rapidly increased to 50,000,000, at fortnightly intervals. He gradually improved. The vaccine treatment was continued until last March, although in February he was well, the diarrhoea and passage of blood and mucus having ceased for a long while, he had gained weight, and was able to go to his work. He is now (May, 1909) perfectly well.

(C) EMPYEMATA AND SUBDIAPHRAGMATIC ABSCESSSES.

Case XV.—The patient, a tall man, seen with Dr. J. H. E. Brock and Dr. F. K. Holman, who met him at Tilbury Docks on his arrival from India, was a mere skeleton, weighing $5\frac{1}{2}$ st., and was so ill and weak

that it was doubtful if he would reach his home at Hampstead alive. I (W. Hale White) saw him a day or two afterwards; he had a hectic temperature, was coughing up considerably over a pint of blood and pus like anchovy paste in the twenty-four hours, and his pulse was feeble and rapid. I was asked to decide whether or not the abscess should be drained, and I gave it as my opinion that the patient was so ill that he stood a better chance of recovery without than with a severe operation. In coming to this opinion I was influenced, not only by his extreme weakness, but also by the fact that examination did not give any clue as to the seat of the abscess, and therefore it might have been a tedious operation to find it, and the amount of blood in the expectoration suggested that incising the abscess might lead to serious hæmorrhage. We accordingly put the patient by a wide-open window and fed him up. Slowly his temperature fell, the expectoration diminished, and he gained weight. After about a couple of months he was able to travel, and spent some months continuing the open-air cure in various seaside places in Devon and Cornwall. Six months after he landed in England he was unrecognizable; he had doubled his weight, but the expectoration was still about 4 oz. a day of pus like anchovy paste. This was examined, and was found to contain *Streptococcus pyogenes longus*. We prepared a vaccine, and on February 15 a dose of 10,000,000 was given, on February 19 one of 50,000,000, on March 7 one of 50,000,000, and on March 27 one of 100,000,000, the dosage being checked by observations of the opsonic index. Under this treatment the expectoration ceased. The patient when last seen, a year after his arrival in England, had, so far as could be made out, nothing the matter with him. There was no expectoration. His weight was $12\frac{1}{2}$ st.—that is 7 st. more than on arrival; he looked perfectly well, and could walk up hills with ease. An examination of the chest with the X-rays, made by Dr. A. C. Jordan, showed that the completeness of the cure was remarkable. The right lung showed no evidence of having had immense quantities of pus (the patient calculated it was 15 gallons since his arrival in England) coughed through it. The right wing of the diaphragm was 2 in. higher than the left, and its excursion, though excellent, was only half that of the left.

Case XVI.—The patient, an old man, suffering from mental disorder, seen with Mr. R. W. Prentice, had had pneumonia four months previously. Since then he had had a hectic temperature, and had been coughing up pus; he had physical signs at the right base. He would not hear of any operation, and, considering his age and his mental condition, we were not sorry. We cultivated *Streptococcus lanceolatus* from

the expectoration and prepared a vaccine. In six weeks two injections of 5,000,000, three of 10,000,000, and one of 50,000,000 were given. He improved greatly under this treatment; his expectoration ceased, his temperature became normal, and he gained weight. When heard of nine months after he was first seen he remained well.

Case XVII.—The patient, a man, aged 60, seen in consultation with Mr. W. Arbuthnot Lane, was short-circuited by him for a duodenal ulcer on December 26, 1908. Two days after the operation he vomited much blood, apparently from the ulcer; some got down into his lungs. Signs of pneumonia appeared at the base of the left lung. He coughed up some pus soon after. His temperature remained raised and he coughed up pus during the last three weeks of January. On January 29 *Staphylococcus aureus* and *Streptococcus longus* were found in the expectoration, and a vaccine was made from each. On February 5 he received a dose of 5,000,000 streptococci, and on February 8 5,000,000 streptococci and 10,000,000 staphylococci. He then went to Bournemouth, under the care of Dr. G. Muspratt, and on February 13 had 10,000,000 streptococci, and on February 16 25,000,000 staphylococci. Directly the vaccine treatment was commenced he began to improve. His temperature slowly fell, his expectoration quickly ceased, and he was soon able to walk about. He is now (May, 1909) quite well and hard at work.

Case XVIII.—The patient, a female, aged 35, had (following a gastric ulcer) a subdiaphragmatic abscess which was drained. *Streptococcus longus* was cultivated from the pus, a vaccine was prepared, the patient had two injections of 5,000,000, and did uninterruptedly well.

Case XIX.—The patient, a female, aged 21, was admitted to hospital for perforated gastric ulcer. An operation was performed by Mr. C. H. Fagge. Subsequently signs of a subdiaphragmatic abscess on the right side developed, and a few hours before operation—for this was to take place—she vomited a quantity of pus. Her temperature came down but soon began to go up again, and pus was discharged by the bowel. From this the *Streptococcus longus* was isolated. A vaccine was prepared from it. After three doses of 5,000,000 each the temperature became normal and the patient was discharged cured. (*See Chart 2.*)

Case XX.—The patient, a female, aged 34, was admitted to hospital on September 30, 1908, for expectoration of pus. She had had pneumonia a month previously. Physical signs and the X-rays revealed an empyema in the right chest in front, extending from the second to the fourth ribs. From the pus she expectorated *Bacillus coli communis*;

Staphylococcus aureus and *Diplococcus pneumoniae* were isolated. Her opsonic index to the colon bacillus was 0.43, to the staphylococcus 1.02, and to the pneumococcus 2.42. Vaccines were prepared from all three, but as the index was normal to the staphylococcus and raised for the pneumococcus the *Bacillus coli communis* vaccine was the only one used. Four doses—viz., 2,000,000, 8,000,000, 20,000,000, and 25,000,000—were given at intervals of a week to ten days. The temperature had

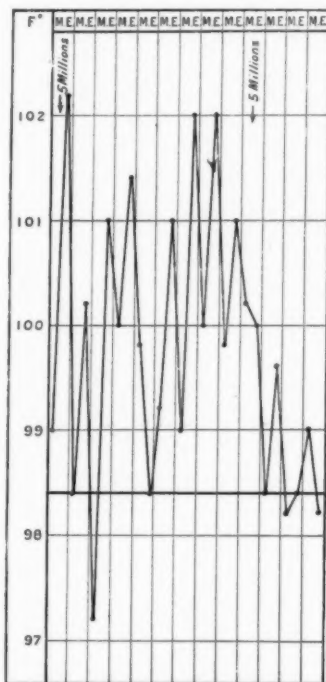


CHART 2 (Case XIX).

Streptococcus longus infection.

been considerably raised for some weeks, but after the first injection it began to fall. After the third it was normal and remained so till her discharge. The expectoration entirely ceased and she gained 2 st. in weight. The index to the staphylococcus and pneumococcus was taken several times and remained constant; the index to the colon bacillus was raised as a result of the injections. The physical signs improved

and an X-ray picture taken before her discharge showed great shrinkage of the cavity of the empyema. During most of her stay in the hospital she was on the open-air balcony day and night. (See Chart 3.)

Case XXI.—The patient, a boy, aged 9, was admitted to hospital for double empyema following double pneumonia. The right empyema was opened on February 6, and the left, after two aspirations, on March 9. A vaccine was prepared from the pneumococcus present in the pus, and

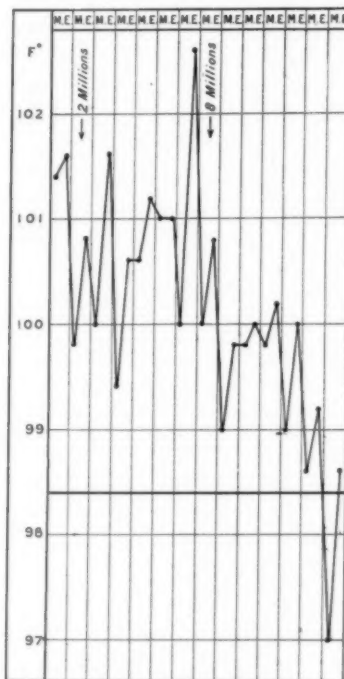


CHART 3 (Case XX).

Bacillus coli infection. Temperature raised for a month before this chart begins.

two doses of 5,000,000 and two of 10,000,000 were administered at intervals of seven days. The boy made an uninterrupted and rapid recovery, gained much in weight, and was discharged cured with the wounds perfectly healed.

Case XXII.—The patient, a man, aged 39, was operated on early in September, 1908, for empyema on the right side. Two pints of pus

were let out. He appeared to be doing well, and the wound closed, but at the end of September the temperature began to rise and continued hectic until November 2, when he was first seen by us. The X-rays and physical signs showed an empyema near the spine at the eighth rib on the right side. Leucocytosis 25,000. Sir Alfred Fripp drained this empyema, letting out a few ounces of pus on December 15. *Staphylococcus aureus* and *Bacillus coli communis* were obtained from the pus. Vaccines of each were prepared, and injections varying from 5,000,000 to 50,000,000 staphylococci and from 5,000,000 to 10,000,000 colon bacilli were injected at average intervals of a week for three months. Although the patient went to the seaside he has not improved much. There is still some discharge from the wound now (May, 1909); he has only gained a little weight, but his temperature is consistently lower, although not normal. He is pale and anæmic. There are no tubercle bacilli in the sputum.

(D) MISCELLANEOUS.

Case XXIII.—The patient, a female, seen with Dr. John Wood, had suffered for a long while from furunculosis, many boils appearing in various parts of the body. A *Staphylococcus aureus* was isolated from one of the boils. A vaccine was prepared, and doses of from 50,000,000 to 100,000,000 were administered. The boils became fewer and less severe, but the improvement was not great, therefore three months after the treatment began a dose of 50,000,000 was followed three days later by a dose of 100,000,000. This produced a rise in the opsonic index to 2.5, accompanied by a rapid disappearance of the last of the boils and striking general improvement in her health.

Case XXIV.—The patient, a male, aged 70, was seen with Dr. C. Ewart for swelling, which, after consultation with a surgeon, was decided to be an inflamed omental hernia. His temperature was raised, and the swelling was very tender. No organisms could be isolated from the blood, and as it was not deemed advisable to incise the tumour the patient's opsonic index to several micro-organisms was estimated, and as that to staphylococcus was low a stock vaccine of *Staphylococcus aureus* was injected. Doses of 5,000,000 and 10,000,000 were given in June, and in eighteen days his temperature became normal, the swelling and tenderness had quite disappeared, and the patient got completely well.

Case XXV.—The patient, a female, aged 45, seen with Dr. N. J. McCaskie for severe osteo-arthritis affecting nearly every joint in her

body and rendering her almost helpless, had over the sacrum a sinus which was several inches long and discharged pus. It had resulted from pressure in spite of careful nursing. The pus was found to contain *Staphylococcus aureus*. The opsonic index to this organism varied between 0.5 and 2.0. A vaccine was prepared. Five injections of it were given in doses of from 5,000,000 to 50,000,000 at intervals of about a week. The sinus was syringed out with peroxide, the pus gradually ceased to be discharged, and the sore improved in every way.

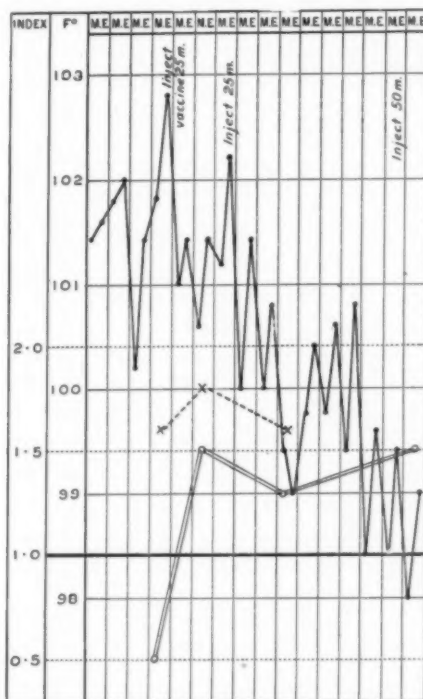


CHART 4 (Case XXVI).

Pneumococcus infection. Dotted line = *Bacillus coli* index; double line = pneumococcus index.

Case XXVI.—The patient, a married woman, aged 27, was confined on April 11, and was first seen by us on April 25 for general septicæmia and septic pneumonia. Cultivations from the vaginal discharge and also from the sputum showed the presence of *Bacillus coli*

communis and pneumococcus. Her index to *Bacilli coli communis* was 1.7 and to pneumococcus 0.5. She was accordingly vaccinated with a vaccine from the latter, three doses of 25,000,000, 50,000,000, and 50,000,000 being used. The improvement was immediate and remarkable, for before this treatment was begun she appeared to be dying. Fourteen days after the first injection the temperature had become normal. (See Chart 4.)

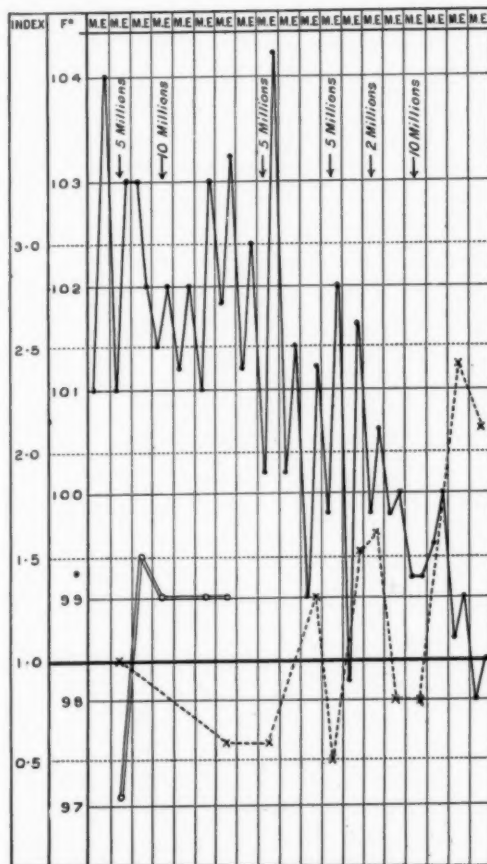


CHART 5 (Case XXVII).

Bacillus coli infection. The temperature had been raised several weeks before the chart begins. Double line = *Staphylococcus aureus* index; dotted line = *Bacillus coli* index.

Case XXVII.—The patient, a married woman, aged 30, was confined on May 22. She had a rigor with a temperature of 104° F. on May 25. Thereafter the temperature fluctuated between 99° F. and 104° F. with frequent rigors until first seen on June 23, when acute nephritis with bloody urine was present. A catheter specimen of urine grew *Staphylococcus aureus* only, and as the index to that organism was only 0·35 two doses of a vaccine prepared from this particular strain were injected. The index rose to 1·5, but the clinical condition remained unaltered. In the meantime *Bacillus coli communis* had been isolated from the vaginal discharge and a vaccine prepared. On July 2 and July 5 5,000,000, on July 7 2,000,000, and on July 10 10,000,000 of coli vaccine were injected. The response was immediate and by July 12 the temperature had returned to normal, and the patient made an uninterrupted recovery and now, a year since, remains in excellent health. (See Chart 5.)

Case XXVIII.—The patient was a married woman, aged 20. This was a severe case of puerperal septicæmia. She was confined on December 7. She had a rigor on December 9. On the following day the uterus was cleared out under an anæsthetic. On the next day large numbers of streptococci were found in the blood. A vaccine was prepared and injections, regulated by the movements of the opsonic index, were given during the next eleven days, at the end of which time the temperature had fallen to 100° F. The patient had improved considerably, the streptococci had disappeared from the blood, and the only sign of infection now remaining was a definite area of parametritis in the right fornix. Three days later the temperature again rose, and there were definite signs of septic pneumonia; no pus could be detected. The patient's strength gradually failed and she died. (See Chart 6.)

Case XXIX.—The patient, a muscular young man, aged 27, had suffered for many years from acne pustulosa, which when he was first seen in May, 1908, completely covered the chest and neck. The face was but little affected. *Staphylococcus albus* was isolated from the pustules and a vaccine was prepared from it. During the next two months six doses of vaccine, commencing with 200,000,000 and gradually increasing to 1,000,000,000, were administered. The response to these injections was most striking, and when the patient left England in the beginning of July the chest was completely clear, the back practically clear, and all of the scar tissues remaining from previous attacks were clearly undergoing absorption.

It will be seen that the first four of the cases of disease of the genito-urinary system were examples of gonorrhœal arthritis, and when we remember how intractable this complaint usually is it will, we are sure, be conceded that our cases did exceedingly well with vaccine treatment. The benefit which followed was so rapid that it was difficult

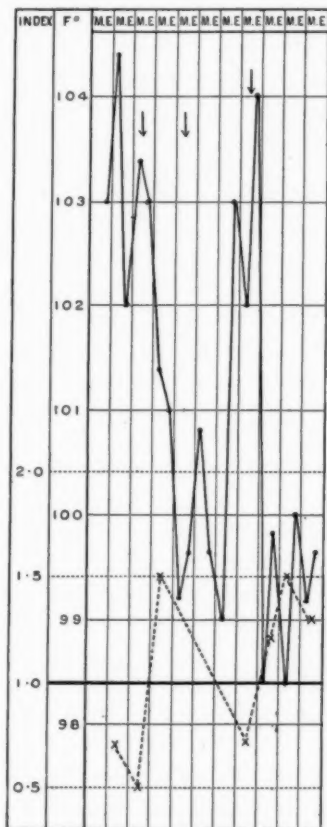


CHART 6 (Case XXVIII).

Streptococcus longus infection.

to doubt that it was due to the vaccine. In the remaining six cases of genito-urinary infection the *Bacillus coli communis* was found in the urine. In Case V the patient was desperately ill when seen, and

had been ill so long that it appeared to us clear that the vaccine treatment saved her life, and it is of great interest to observe how quickly improvement followed the first injection. Case VI was very ill, and had had considerable pyrexia for over three weeks; yet two days after the first injection of coli vaccine the temperature was normal. He was treated with vaccines prepared from both micro-organisms found in his urine, but the *Bacillus coli communis* was probably the most active for harm, for it was after the injection of this that the temperature at once fell. Case VII was a mild case of cystitis, but it is of interest that she benefited in spite of her extreme age. Case VIII did extremely well in spite of the long duration of the cystitis. In Cases IX and X the vaccine treatment appeared to do no good. Why this was so in Case IX is not quite clear, but it is to be noted that although her urine contained enormous quantities of the colon bacillus there was no pus, so that she can hardly be said to have had cystitis or pyelitis in the ordinary sense of the terms. Case X was so ill from his heart disease that he died before any conclusions could be drawn as to efficacy of the treatment. Therefore out of ten genito-urinary cases, in the four due to the gonococcus the improvement was rapid after vaccine treatment, and of six in which *Bacillus coli communis* was the cause of the symptoms from which the patient suffered four did extremely well, and in two cases—viz., V and VI—the treatment, in our opinion, saved the patient's life.

Turning next to the alimentary cases, Case XI, in which the severe attacks of dyspepsia accompanied by fever were associated with and apparently due to *Bacillus coli communis* in the stomach, was very remarkable. Although an occasional colon bacillus may be found in stomach contents, yet in this patient a loopful of the stomach washing contained millions of *Bacillus coli communis* in pure culture, and it would appear that this case is an example of a hitherto unrecorded cause of severe dyspepsia. We may mention that we sought for these bacilli in another case of unusual dyspepsia, but without finding them. That they were the cause of the dyspepsia in Case XI appears extremely likely from the fact that as the patient improved under the vaccine treatment these bacilli almost completely disappeared from the stomach contents, and that this treatment helped her very considerably appears plain, for no previous treatment had benefited her at all. The remaining three gastro-intestinal cases were all examples of ulcerative colitis, in which *Bacillus coli communis* was found in the fæces. Whatever may have been the original cause of the ulcerative colitis, that this

bacillus was, when we saw the patients, the actual cause of the chronic inflammation of the large bowel is highly probable, for in each case the patient's blood contained specific agglutinins, easily demonstrated in high dilutions, against the particular *Bacillus coli communis* which we recovered from the fæces and utilized in the preparation of each vaccine. Moreover, in each case the opsonic index to the *Bacillus coli communis* recovered from the fæces showed remarkable variations from day to day, ranging from 0.5 to 2.5. These three cases encourage us to hope that in the future much good may follow the treatment of this intractable disease by vaccines, especially if combined with milk soured with lacto-bacilline, and perhaps the administration of cyllin.

Eight cases fall under the heading of empyemata and sub-diaphragmatic abscesses, and we are well aware that both these disorders often recover without vaccines, but in Case XV the diminution of the expectoration, which had lasted many months, was so prompt after the giving of vaccines that it appeared to be due to them. The same was true of Cases XVI and XVII, XIX and XX, and our impression derived from these eight cases is that the healing of these abscesses, usually so tedious, can often be accelerated by the administration of the appropriate vaccine. The only case in which it appeared to do no good was Case XXII.

Among the miscellaneous cases only Nos. XXVI and XXVII call for comment. No. XXVI had very severe puerperal septicæmia with a large amount of septic pneumonia. She was so near death's door before the vaccine treatment was commenced that our strong impression is that it saved her life, and the same is true of No. XXVII; until the vaccine treatment was adopted her temperature was raised for six weeks and she had remained consistently very ill; after vaccination the temperature fell and she improved in every way. In all the cases recorded in this paper, unless otherwise stated, the vaccine treatment was the only special treatment adopted.

We have given brief details of each case occurring since January 1, 1908, in which we have employed vaccines, recording failures as well as successes, in order that those who are present may form their own conclusions. For our own part, we believe that in not one of these twenty-nine cases did the vaccines do any harm, and in the great majority it appeared to us that distinct benefit followed from them; in some, indeed, they, in our opinion, saved the patient's life.

DISCUSSION.

The CHAIRMAN (Dr. Ince) said the paper showed how increasingly hopeful in the cure of disease medicine and surgery had become. They could never hope to prevent eventual death, but most people suffered to some extent during their lifetime, and this could be very greatly relieved. Some of those present had had some experience of the influence of vaccine treatment, and the Section would be glad to hear the results of their observations. Vaccine treatment seemed to be opening up a new form of combating disease; certainly the paper showed a good deal of devotion to the subject, and the Section would cordially express its thanks to the authors.

Dr. HORT read the following communication: Notwithstanding the favourable verdict of the distinguished authors of to-day's paper, and in spite of many and varied successes claimed by the vaccine school, I am heretic enough to confidently indict the system of bacterial vaccine-therapy as practised in this country to-day. The method is, I am convinced, so built up of misconception and so embedded in assumption to which at present we are not entitled that, unless most carefully restricted to its proper sphere, it will, sooner or later, inevitably become discredited. I do not, of course, extend indictment of the method to its adoption in such relatively unimportant affections as those for which it is often and quite legitimately employed. In these, characterized for the most part by trifling tissue change, the method, as at present understood, has, as we have heard, a demonstrably very useful field. My concern is not to belittle its success in these nor in scattered cases of more serious disorder. But I do protest that if real advance is to be made in the art of immunization we must take a much broader view of infective disease than holds to-day, and not, by our practice, confine ourselves to its purely bacterial aspect. The fundamental error of exponents of bacterial vaccine-therapy as now followed is, I am convinced, this—that the intracellular factor in disease has been forgotten or ignored. We assume, that is, that the cell, as a unit, is only concerned, apart from its own nutrition, in antagonizing extramural foes, such as bacteria and their products, and that enemies within its gates, such as cell-ferments and the toxic products of their activity, have no existence. Believing that bacteria are the primary and essential cause of diseases ostensibly associated with their presence, we assume that toxæmia is all bacterial. Hence we imagine that by first provoking, and then conveying to the field of strife, antibodies to bacteria and their toxins we are fulfilling the whole duty of man. Recent research in cellular pathology has made clear two well-recognized truths that should have saved us from error: Cells and their ferments of one species are poisonous to those of another, and would be and are poisonous to themselves under certain conditions, unless constantly restrained by their own anti-ferments. The second truth is this: The products of morbid-cell activity are highly toxic, and auto-toxæmia of cell origin, which may be set going sometimes by bacterial action, is certainly not identical with bacterial toxæmia. In a

word, we do not recognize that in any infection we may be called upon to exert both cellulo-tropic and bacterio-tropic restraint. No bacterial body, nor bacterial antitoxin, nor bacterial opsonin can possibly enable cells already damaged to restore the condition of immunity to intrinsic morbid cell change on which effective cell life depends. Nor can they neutralize toxic cell derivatives. The utmost they can do is to prevent further damage from extrinsic forces, such as bacteria and their toxins only. We have no right to assume that intrinsic cell change may not as often precede infection as follow it. If to bacterial vaccines were added vaccines made of emulsions of cells and their products, great possibilities would be opened, both as regards prophylaxis and cure, but so far this has been neglected. Nature, fortunately, when she cures infection, considers all the factors involved, for she converts both tissues and bacteria into vaccines and insists on both cellular and bacterial restraint. Unless we, too, do likewise we are bound often to fail whenever cell response is in serious demand. Happily, we have in auto-vaccination an admirable substitute. Whenever practicable, this is the safest, simplest, cheapest method of vaccination to employ, and, being a strict imitation of the best possible model of immunity production to employ, is also the most scientific. It will eventually find wide acceptance, leaving emulsions of dead bacteria to a useful but limited sphere. Already it has much to its credit, and if severe cases of tuberculous empyema, pulmonary tuberculosis, tuberculous epididymitis, sinus, intractable fistula, acne, psoriasis, eczema, ulcer, abscess, &c., cured or arrested without the use of tuberculin or other extraneous vaccine are any gauge of its efficacy, you will admit the solid foundation of fact. In brief, as at present employed, vaccine-therapy asks for half and expects the whole. Rational immunization, not content with half, demands the whole, and, if the power of response is there, it is forthcoming.

Dr. McCULLOCH said that he began to deal with serum-therapy just ten years ago in India in streptococcic infections, chiefly puerperal, where antitoxin was invariably successful, without the isolation of the responsible micro-organism (although more than one variety was concerned) by estimations of the opsonic indices to each variety. Clinical evidence had alone to be relied upon. He believed Sir Almroth Wright admitted the inadequacy of the term "vaccine," in reference to the suspensions of dead organisms used in emulsion, for injection. The Jennerian "vaccine," as he understood it, was a suspension or emulsion elaborated *in vivo*, and therefore more related to an antitoxin prepared vicariously than it was to a mere suspension of dead organisms in normal saline solution. Dr. Hort, he thought, had very rightly drawn attention to the occurrence of *auto-vaccination* in the infectious processes, whether these were simple or complex. He had himself directed attention to this in a contribution made to the *Lancet*, January 26, 1906. It is conceivable that on the injection of a so-called vaccine an elaboration of antigens, followed by that of a true vaccine, must take place in the tissue fluids, much in the same manner as *secretin* follows upon the elaboration of *pro-secretin* in the stomacic and duodenal mucous tissues. He suggested that it would be more scientific to

adopt a similar nomenclature to that of Professor Starling by speaking of these suspensions of organisms as *pro-vaccines*. He noticed that the author of the paper had been guided in selecting his remedial suspensions, as he found a low index to one or other of the organisms involved, and this was doubtless a sound procedure in the majority of cases. He had, however, found that extreme indices—that is, very low or very high ones—were unfavourable periods for the injection of tuberculin R., and the same might apply to the injection of “vaccines.” Each variety of toxic organism seemed capable of evoking a peculiar range of response, though this was doubtless governed by other factors. Staphylococccic opsonic charts in their extremes of variation reminded him of the thermic chart of an intermittent ague, while that of tubercle with moderate dosage fluctuated much less. He thought that the different types of ranges of charts might be worked up by pathologists with advantage.

Dr. EYRE, in reply, said his part of the work had been largely confined to the technical details in the laboratory, which, although valuable in themselves, did not appear very much. Vaccine treatment was supposed to depend very much on intuition, and the results were not always very obvious or very intelligible. A remark had been made with regard to the method by which they sorted out the organism which was responsible for any given infection, and the way in which the vaccine had been used against that particular germ. In that connexion he wished to say that, though they had made their details as brief as possible, in most of the cases several organisms were used in testing the resisting power of the blood, and in many of the cases those estimations had been made at frequent intervals throughout the course of the disease. In one case, for instance, they gave vaccines of the *Bacillus coli*, which was present, and it was found that the index to the pneumococcus, which was also present, was consistently raised throughout the illness—that is, the patient very successfully immunized herself against that particular germ—and it was only when the colon bacillus was brought into the field that it was found that the powers of resistance to that organism were low; so that, although the patient was infected at the time with both organisms, only one vaccine was needed for the purpose of treatment. That the method of treatment was correct was evidenced by the very rapid response which she made to the injections of the colon bacillus vaccine. In another case there were three organisms present. In two instances the indices remained fairly constant; to one organism it was high, to another normal, and to a third it was low, and their efforts were directed against that germ. But he did not wish for a moment to express the opinion that a low index to a germ was an absolute indication for treatment with vaccines of that organism, or that a very high index was an indication against the treatment. They desired to find out whether a patient's resistance to a given germ was remaining steady from day to day. A patient might have a high index to an organism on one day, while on the next day it might be quite low. Under those conditions a vaccine was useful, as results had shown. It was also found that in the cases where vaccines were going to do good they did good quickly. In many of the cases in which they had given vaccines only three or four doses

had been given. Cases which did not respond well in the early stages were not the cases for the continuance of the method, or perhaps they were cases which would do as much good without the vaccine treatment at all. Stock vaccines were, as a rule, inferior to vaccines prepared from the infecting organism. Tubercle was an exception. If one was lucky, one might isolate a pure cultivation of tubercle bacilli in four to six weeks, and one might make a special tuberculin from that. But they were not usually lucky, and the time occupied in preparing the tuberculin might be months. Luckily, tuberculosis being such a chronic condition, and the bacilli differing so little from each other, the tuberculin which had been prepared by various manufacturers, according to Koch's formula, was useful for all tubercular conditions. But for the acute bacterial infections the vaccine could usually be prepared in thirty-six hours, and sometimes, by working almost continuously, one could get a vaccine in twenty-four hours.

The Action of Digestive Ferments upon each other.¹

By WILLIAM BAIN, M.D.²

It is obvious that any pancreatic preparations given by the mouth, unless protected by keratin, must be subjected to the action of the gastric juice. With this fact in view the investigation now reported was undertaken. At the outset it was sought merely to ascertain the action of an artificial gastric juice upon some digestive preparations used for therapeutic purposes. Later, however, the investigation was extended. Attention in this phase of the work was directed towards estimating approximately the time taken by the gastric juice to inhibit or destroy enzymes. Per contra, the effect of trypsin on pepsin was included in the research.

LITERATURE.

Bidder and Schmidt [2] stated that the fat-splitting ferment is only active in alkaline or neutral solutions; certainly not in acid solutions.

Grützner [7] also pointed out that the fat-splitting ferment is destroyed by acids. If he kept his glycerin extracts of the pancreas for some days they became acid and then inactive.

¹ Read at the meeting of April 6, 1909.

² From the Physiological Department, King's College, London.

Kühne [9] stated that trypsin is destroyed by pepsin, but that trypsin does not affect pepsin.

Ewald [4] confirmed Kühne's observations.

Engesser [3], whose pancreatic preparations were used by Ewald, doubted these conclusions, and stated that pancreatinine does not lose its proteolytic action after digestion with pepsin in 0.5 per cent. hydrochloric acid.

Mays [12] stated that pepsin favoured the deleterious action of weak acids on trypsin.

Langley [10] found that pepsin and trypsin are destroyed by each other—that trypsin acts on pepsin by increasing the inhibitory action of the alkali. He further found that rennet is destroyed by trypsin and that pepsin weakens diastase.

Falk [5] stated that pancreatic juice and papain do not act on ptyalin and diastase; and that pepsin is without influence on invertin. The latter observation is confirmed by Richet [13] and Szabo [14].

Marcus and Pinet [11] found that papain does not affect diastase or ptyalin.

Baginsky [1] stated that pepsin destroys trypsin.

Fermi and Pernossi [6] found that pepsin has no perceptible action on trypsin.

V. Harley [8] found that trypsin is destroyed by pepsin-hydrochloric-acid, but pepsin is not destroyed by trypsin. Papain is also destroyed by pepsin, but papain and trypsin do not destroy each other.

Wroblewski, Bednarski, and Wojczynski [15], in a recent paper, state that pepsin acts on trypsin, but not so strongly as to destroy or inhibit the trypsin completely in a short time; that it helps the detrimental action of acids. In ascertaining the action of trypsin on pepsin they used egg white, estimating the nitrogen. They found that 0.5 per cent. sodium hydrate destroyed pepsin, and that 0.1 per cent. weakened it. Even without alkali the trypsin solution acts on pepsin, but when the alkali is present the inhibitory influence is increased. They further found that rennet is not destroyed by pepsin; that trypsin does not act on diastase, but pepsin does so and increases the unfavourable action of acids.

It will be seen that past work has yielded divergent and often conflicting results.

The experiments now to be described were chiefly concerned with the action of an artificial gastric juice upon the proteolytic, amylolytic and

lipolytic ferments of the pancreas. We shall consider first the proteolytic ferments.

The method adopted for estimating the effect of pepsin on trypsin was that described by Roaf. It consists in staining fibrin with congo red. When the fibrin is attacked by the ferment the congo red is set free and diffuses throughout the solution. The activity of the ferment is gauged by the depth of the pink colour of the fluid. The preparations used in these experiments were pancreatinine; liquor pancreaticus; holadin; liquor trypsin Co; papain and liquor papain. The latter two are, of course, vegetable ferments. These preparations are not equally active, and as regards the liquid ones their activity is greatest within a short period of their preparation.

The experimental method was as follows: The artificial gastric juice—the standard used throughout—consisted of 1 gm. scale pepsin (Fairchild), 6 c.c. hydrochloric acid, specific gravity 1.19, to 1000 c.c. of distilled water. 200 c.c. of this solution was taken, and to it was added a definite quantity of one of these preparations; the amount being the dose usually given to a patient—for example, 8 c.c. of liquor pancreaticus was added to 200 c.c. of the pepsin solution. The quantity of gastric juice secreted during an average meal is probably about 200 c.c.; therefore that amount of the pepsin-hydrochloric-acid solution was taken so that the experiments might be roughly comparable to the natural process. The flask containing the 200 c.c. of the pepsin solution, plus 8 c.c. of liquor pancreaticus, was plugged with cotton-wool and placed in the incubator for times varying from one to three hours. The incubator was kept at 36° C., and the flask was shaken at intervals. To the solution was then added the known amount of sodium carbonate necessary not only to neutralize the acid but to convert the solution into one containing 0.5 per cent. of the alkali. Half a gramme of congo red fibrin was added to the alkaline solution, and the flask returned to the incubator for about an hour. If the action of the ferment was inhibited or the ferment destroyed, the solution remained colourless or was very slightly tinged. In every instance control experiments were made which demonstrated that the original pancreatic preparations were active. The results are tabulated below.

To prevent repetition in the tables, it may be stated that in all these experiments 8 c.c. of the liquid preparations, $\frac{1}{2}$ gm. of pancreatinine and papain, and 9 gr. of holadin were respectively added to the 200 c.c. of the pepsin-hydrochloric-acid solution.

TABLE I.

Digestive preparation added to pepsin-hydrochloric-acid solution	Quantity of pepsin- hydrochloric-acid solution	Time the mixture of enzymes was kept in incubator	Subsequent action on congo red fibrin, after the mixture was made alkaline
Liq. trypsin Co ...	200 c.c.	3 hours	No action
Liq. pancreaticus ...	200 c.c.	3 hours	No action
Papain ...	200 c.c.	3 hours	No action
Holadin ...	200 c.c.	3 hours	No action

TABLE II.

Digestive preparation added to pepsin-hydrochloric-acid solution	Quantity of pepsin- hydrochloric-acid solution	Time the mixture of enzymes was kept in incubator	Subsequent action on congo red fibrin, after the mixture was made alkaline
Liq. trypsin Co ...	200 c.c.	2½ hours	No action
Liq. papain ...	200 c.c.	2½ hours	No action
Pancreatinine ...	200 c.c.	2½ hours	No action
Holadin ...	200 c.c.	2½ hours	No action

TABLE III.

Digestive preparations added to pepsin-hydrochloric-acid solution	Quantity of pepsin- hydrochloric-acid solution	Time the mixture of enzymes was kept in incubator	Subsequent action on congo red fibrin, after the mixture was made alkaline
Liq. pancreatinine ...	200 c.c.	2 hours	No action
Liq. trypsin Co ...	200 c.c.	2 hours	No action
Holadin ...	200 c.c.	2 hours	No action
Papain ...	200 c.c.	2 hours	No action

TABLE IV.

Digestive preparations added to pepsin-hydrochloric-acid solution	Quantity of pepsin- hydrochloric-acid solution	Time the mixture of enzymes was kept in incubator	Subsequent action on congo red fibrin, after the mixture was made alkaline
Pancreatinine ...	200 c.c.	1½ hours	The solution was pale pink
Liq. trypsin Co ...	200 c.c.	1½ hours	The solution was pale pink
Holadin ...	200 c.c.	1½ hours	The solution was pale pink
Papain ...	200 c.c.	1½ hours	The solution was pale pink

After one and half hours' incubation the solutions were slightly pink, thus showing that the trypsin was not completely destroyed. Where the flasks were incubated for one hour the solutions were deep pink, but the depth of colour was less than in the control experiments. It is thus evident that a portion of the trypsin is destroyed or its action lessened during an hour's digestion in the pepsin solution.

The effect of the pepsin solution on the digestive preparations at room temperature was next tried. The solutions were allowed to stand in the laboratory for three hours, then made alkaline; fibrin added and incubated. Result: Digestion did not proceed nearly as briskly as in the control experiments. Similar experiments with 0.27 per cent. hydrochloric acid (without pepsin) gave a like result, thus showing that the acid weakens the proteolytic power of trypsin.

A pathological gastric juice was devised. It consisted of equal parts of the standard pepsin solution and distilled water, to which was added 0.1 per cent. lactic acid.

Experiments were carried out with this solution similar to those with the standard pepsin solution.

After three hours' digestion in the pathological fluid the proteolytic power of trypsin was destroyed.

The Effect of Trypsin on Pepsin.—To 200 c.c. of a 0.3 per cent. sodium-carbonate solution was added $\frac{1}{4}$ gram. of pepsin and 8 c.c. liquor trypsin Co. The flask was incubated for three hours, and then a known quantity of hydrochloric acid was added to make the solution one containing 0.2 per cent. of the acid; $\frac{1}{2}$ gram. of fibrin was added and the flask again incubated for an hour. Result: The fibrin was unaffected.

Without entering into further experimental details, it may be stated that trypsin and papain destroy each other and that papain acts on pepsin.

As regards the amylolytic ferment of the pancreas, the method adopted for ascertaining its activity was a modification of that described by Wohlgemuth. A series of test tubes were taken each containing a decreasing quantity of the ferment. To each tube was added 5 c.c. of a 1-per-cent. solution of starch and the tube at once placed in iced water. When all the tubes were prepared they were put in the incubator for an hour. On removal from the incubator they were again placed in iced water. They were then filled to the brim with water and one drop of a one-tenth normal iodine solution added. If the ferment was active, no colour was produced; if the ferment was destroyed, the colour was blue.

The digestive preparations used were liquor trypsin Co, liquor pancreaticus, and holadin.

A flask containing 200 c.c. pepsin-hydrochloric-acid solution and 8 c.c. liquor pancreaticus was incubated for an hour. The solution was then neutralized and rendered very slightly alkaline; 1 c.c. of this solution was added to 5 c.c. of the starch solution and incubated for an hour. Result: No action on starch. Larger quantities of the ferment solution were added to the starch solution, and the same result obtained. Similar results were also obtained with other pancreatic preparations, thus showing that the amylolytic ferment is completely destroyed during an hour's digestion in the pepsin-hydrochloric-acid solution.

To ascertain the action of the artificial gastric juice upon the lipolytic power of the various pancreatic preparations, the lipolytic ferment was allowed to act upon an emulsion of olive oil, and the amount of fat disintegration estimated by the titration of the free fatty acids. The emulsion was prepared in the following way: The amount of acidity of the olive oil was ascertained by titrating 5 c.c. of it in 30 c.c. of alcohol with decinormal caustic potash, phenolphthalein being used as the indicator. The stock emulsion was made by adding the calculated amount of caustic potash to the olive oil. The preparations used were holadin, Grubler's steapsin and glycerin extract of the pancreas prepared in the laboratory. One experiment will be described in detail:—

Two flasks—one containing 200 c.c. pepsin-hydrochloric-acid solution and 8 c.c. of steapsin, and the other containing 200 c.c. distilled water and 8 c.c. of steapsin—were placed in the incubator for an hour. The solutions were then neutralized with caustic potash, phenolphthalein being the indicator and made of equal volume. Two small bottles, each containing 10 c.c. of olive-oil emulsion, were taken. To No. 1 was added 10 c.c. of the neutralized pepsin-steapsin solution. To No. 2 was added 10 c.c. of the neutralized steapsin solution. These were placed in the incubator for six hours. Finally, to each was added 40 c.c. of absolute alcohol, and the free fatty acids titrated with decinormal caustic potash. The alcohol was added to prevent hydrolysis. Control experiments were made.

The result of one experiment will be given in tabulated form. In this experiment the laboratory extract of pancreas was used: 10 c.c. of it was added to 200 c.c. pepsin solution and digested for an hour; 10 c.c. of the neutralized solution was added to 10 c.c. of the olive-oil emulsion and incubated for 22 hours.

TABLE V.

	Time in incubator	Acidity before incubation estimated by titration with one-tenth normal KOH.	Acidity after incubation estimated by titration with one-tenth normal KOH.	Difference
10 c.c. neutralized pancreas and pepsin solution in 10 c.c. olive-oil emulsion	22 hours	0.8 c.c.	2.0 c.c.	1.2 c.c.
10 c.c. neutralized pancreas extract in distilled water and 10 c.c. olive-oil emulsion	22 hours	1.2 c.c.	4.5 c.c.	2.3 c.c.

This shows that the injurious influence of pepsin-hydrochloric-acid on the lipolytic enzyme of the pancreas is not so marked as in the case of the other enzymes, and accords with the fact which has now been established that gastric juice itself contains lipase. Nevertheless, incubation of the pancreatic lipase with pepsin-hydrochloric-acid for one hour reduced its activity by about one-half.

So far as it is safe to draw conclusions from experiments *in vitro* to the occurrences taking place in the alimentary canal, the point of practical importance arising from the work is that in the administration of pancreatic preparations by the mouth measures should be adopted to protect them from the deleterious influence of gastric juice, especially if one wishes to favour the intestinal digestion of protein or of starch. The danger may be minimized by giving the pancreatic preparations at a time when the stomach is emptying itself rapidly into the duodenum; if given from half an hour to an hour after a meal this will be ensured, and the length of time the pancreatic preparations will remain in contact with gastric juice will thus be lessened. In pathological cases (for instance, in cancer), where the gastric juice is inactive owing to the absence or lessening of hydrochloric acid, the deleterious influence of gastric juice may be minimal.

SUMMARY OF RESULTS.

(1) After two hours' digestion in pepsin-hydrochloric-acid solution at a temperature of 36° C. the tryptic action of medicinal doses of various pancreatic digestive preparations is entirely destroyed.

(2) During one and a half hours' digestion the tryptic action is not completely destroyed.

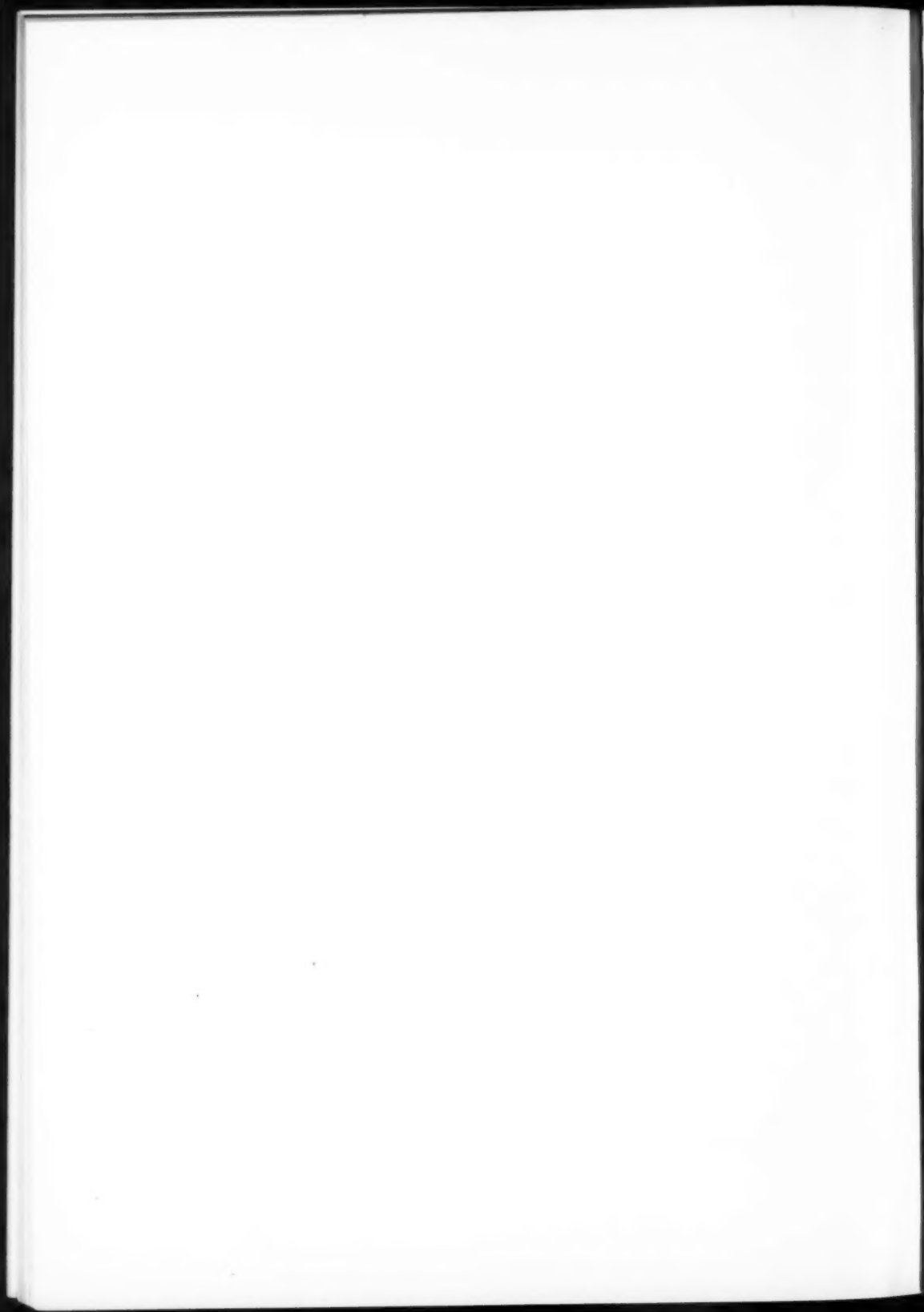
- (3) Trypsin destroys the activity of pepsin and papain.
- (4) Amylolytic action is destroyed by one hour's digestion in the standard pepsin-hydrochloric-acid solution.
- (5) The lipolytic enzyme retains about half its normal activity after an hour's digestion in the pepsin-hydrochloric-acid solution.

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PROCEEDINGS OF THE ROYAL SOCIETY OF MEDICINE, Vol. II, 1909



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(Prepared by Mr. A. L. CLARKE)

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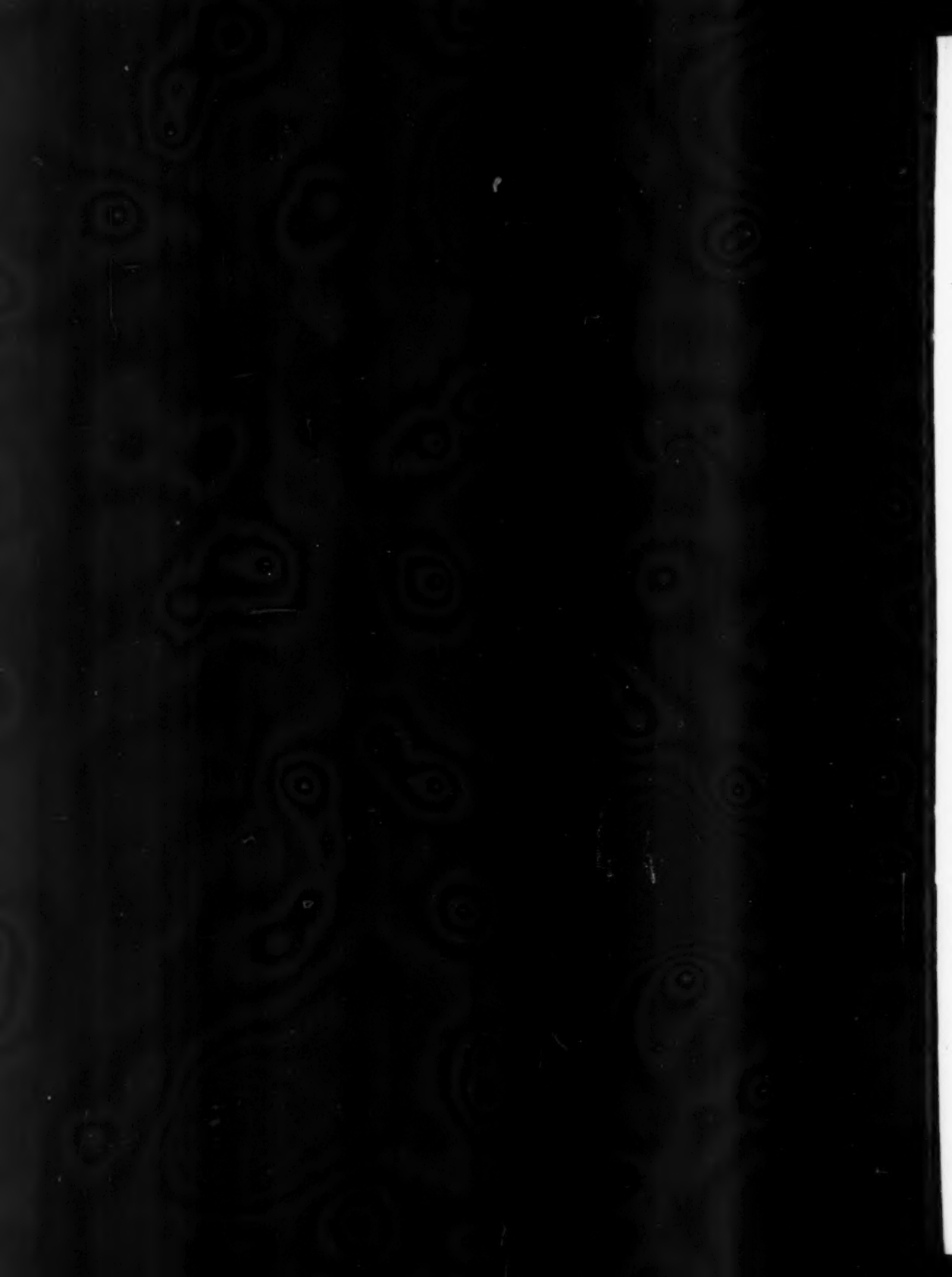
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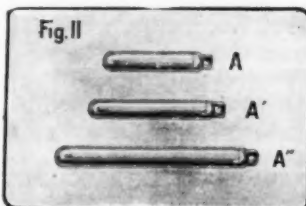
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